ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 44. JUNE, 1935. No. 2.

XXXI.

THE OPERATIVE TREATMENT OF NASAL SINUS DISEASE.*

EDWARD CECIL SEWALL, M. D.,

SAN FRANCISCO.

A discussion of the operative treatment of nasal sinus disease necessitates the correlating of various technical endeavors, some as yet unpublished, and the evaluating of methods and hoped-for improvements with which I have been occupied since the publication of my original article entitled "Fronto-ethmo-sphenoid Operation Under Local Anesthesia," in the Archives of Otolaryngology, in November, 1926, I am inclined to quote from that article as follows, because what was said then needs even more emphasis today:

"Indications for operation. The operative treatment of sinus disease is a subject that I feel should be approached with considerable caution. I am proposing a rather radical line of procedure that I feel may add to our efficiency in handling certain cases. Still I do not wish to leave the impression that I am applying these measures except after careful study or when former unsuccessful operative work would seem to make their applicability advisable. I have gone into the question of diagnosis frankly, but must make the point plain that the diagnosis arrived at does not condemn the patient to operative measures. It becomes a matter of the nicest judgment on the part of a well trained surgeon, probably with the invaluable assistance of the internist, to determine whether the sinuses should be operated upon. A rubric of the operative indications would be only about as useful as those that have been compiled to tell us when to operate upon a chronic running ear. Many people with apparently excellent health are sufferers from infected sinuses. The hawking and spitting that is so commonly heard, and the mucopurulent expectoration seen, are evidence of the widespread distribution of sinus disease.

*Presented before the meeting of the Western Section of the American Laryngological, Rhinological and Otological Society at Los Angeles, January 19, 1935.

MEDICAL LIBRARY

While admitting that many people seemingly well are carriers of this infection, it is obvious that they are apparently in good health in spite of the sinusitis. It is a menace constantly carried about, and often proves to be the vulnerable heel of Achilles, leading to the establishment of some important vital disorder. The inability to discover the true focus, the removal of the wrong focus or the incomplete treatment of the right focus, have brought a certain opprobrium on the focal infection theory. Incomplete treatment of the right focus is practiced so commonly in connection with sinus disease that the specialists must take the onus of the lack of understanding on the part of many of the medical profession of the importance of sinus infection. These men have conscientiously and repeatedly referred patients for the elimination of nasal infection. Failure even after nasal sinus operation to cure the disorder in which the internist was interested, very frequently has been interpreted as indicating improper diagnosis. The use of the method I have advocated of gathering the nasal and postnasal discharge from the patient on pieces of cloth will show the internist himself whether the results he has asked for have been achieved.

"Ordinarily, before operation on the sinuses is to be considered, all other factors that may be contributing to the trouble should be corrected; climate has been given especial prominence. Whatever therapeutic measures have been found useful may be employed.

"Constructive surgery, when indicated, including submucous resection, tonsillectomy, and adenoidectomy should be given precedence and followed by sufficient time for accomplishment of results before the destructive surgery of the sinuses is advocated."

In presenting my present operative plan, it may be said that the intricate technical developments have followed naturally upon the introduction of a bloodless technic. This was accomplished by ligation of the vessels supplying the operative field, under local anesthesia secured largely by blocking the nerves before or at their entrance to the field. Sinus surgery may well be divided into a pre- and a post-ligation period. The literature of the former contains many descriptions of the bloody operative field where all nicety of technic was impossible. General anesthesia was then, and often still is, used routinely in the performance of sinus surgery, via external approach.

TECHNIC OF THE FRONTO-ETHMO-SPHENOID OPERATION, UNDER LOCAL AND BLOCK ANESTHESIA.

Preparation of the patient:

Analgesia.—Sodium amytol is given by mouth; scopolamin and morphin hypodermically. Scopolamin gr. 1/150 to 1/100, in my experience, has not caused the nervous reaction described by some.

Anesthesia.—Cocain is used locally in the nose by topical application. A fine wire applicator wound with a small bit of cotton is moistened with adrenalin and dipped lightly into flake cocain. Only

what dissolves is taken. This gives a strong, fresh solution, not cocain "mud." The nose is never packed with cocain saturated materia because the danger of absorption is a real one. Novocain is injected superficially and deep in the skin. A long needle is then passed into the orbit to block the branches of the fifth nerve, the supra-orbital, supratrochlear and the ethmoidals.

The Skin Incision.—The skin incision follows the curve at the inner angle of the orbit, extends below the unshaved eyebrow outward not farther than the supraorbital notch—artery thus avoided—and downward onto the nasal process of the maxillary bone. A length of three or four cm. ordinarily gives enough room because of the flexibility of the soft parts. However, as the scar becomes practically invisible, as much room may be taken downward as required. Ordinarily the frontal sinus can be cared for through this incision. There are plenty of cases, however, where the frontal sinus is so large—extending to the external angular process of the frontal bone—that it cannot be adequately handled through this cut, nor will such a sinus drain properly into the nose as is expected according to the classical technic. In such cases I use a supplementary incision at the outer angle of the orbit underneath the eyebrow, 3 to 4 cm. long as required. This incision permits attention to the lateral part of a large frontal sinus and will be discussed later.

Opening Through the Bone.—The periosteum is elevated, exposing the antero-inferior wall of the frontal sinus, and the frontal process of the maxillary bone. The lacrimal sac is partly raised from its fossa and the periosteum from the anterior few millimeters of the lamina papyraceæ. The anterior ethmoid artery and nerve are perhaps seen in the depth as the periosteum is raised from the lamina.

The lower end of the bony window is cut through with a sharp chisel. This cut is made first while the bone is attached and firm as possible. Care is taken not to cut or injure the underlying nasal mucosa which is to be preserved to form a frontal flap. The upper end of the window is made by entering the frontal sinus at the inner angle of the orbit. The medial limit of the window is carefully determined by study through the frontal sinus opening and through the nose. This bone incision should be as central as possible and still avoid thick septal and frontal bone. It is made with a sharp, flat chisel.

The fourth side or lateral wall of the bone fractures easily at the thin laminal edge. The bone as it is removed is carefully separated from the valuable nasal mucosa. The inner surface of this square of bone is pitted by part of the anterior ethmoid cells. The bone is

thus quickly removed and admittance gained to the frontal, ethmoid and sphenoid sinuses.

Control of Bleeding.—It has long been recognized in general surgery that adequate control of bleeding is essential to proper surgical work. It was apparent that the application of this fundamental surgical principle was imperative if nasal surgery was to progress. In 1920, I managed to ligate all of the vessels of consequence that supplied the nose and accessory sinuses. These ligations, limited exclusively to the vessel supplying the operative field, are accomplished through the operative incision and in the immediate neighborhood of the surgical field.

Prior to these ligations the rhinologic surgeon depended upon the local application of cocain and adrenalin for ischemia. The complicated and difficult technic herein described can only be carried out under the complete control of bleeding. The vessels that require ligation in the work under discussion, the fronto-ethmo-sphenoid operation, are the ethmoid arteries in all cases and the sphenopalatine artery when it is necessary to remove the floor of the sphenoid sinus. I have recently used the high frequency coagulating current in handling these vessels. This may be found to simplify the technic materially if reliance can be placed on such control of these rather large vessels.

The usefulness of ligation in the ethmoid vessels, where they are at fault, in severe, otherwise uncontrollable nasal hemorrhage, is advocated. The usual last minute stampede to control the nasal bleeding from this source by ligation of the external carotid artery is misdirected. These vessels are branches of the internal carotid artery via the ophthalmic artery.

The ethmoid arteries differ anomalously in size, number and location. They are ligated, as originally described, by separating the orbital periosteum from the frontal bone and the lamina papyracea, and passing the ligature around the vessel as it passes medially through its foramen covered by orbital periosteum. They are then cut on the nasal side of the ligatures.

Treatment of the Frontal Sinus.—The frontal sinus has been opened in forming the bony window as described. Even if the orbital sinus is not diseased the removal of much of its floor is necessitated because the ethmoid cells so often extend orbitally beneath it. The frontal sinus is therefore always included in this operation and the provision for its drainage must be provided. This responsibility gives pause to the thoughtful surgeon to weigh carefully all considerations before operating.

The entire floor of the frontal sinus is removed. It forms much of the orbital roof. It is impossible to accomplish this through the original incision without undue trauma to orbital contents when the frontal sinus extends laterally to an extreme degree. Therefore, in such cases, a counter incision as already described, is made external to the eyeball. This supplements the original incision to permit the complete removal of all of the floor of an extremely large sinus. It serves the added purpose of affording dependent drainage for the outer part of a very large sinus which could never drain successfully over the curve of the orbital contents and down into the nose, as required by the single incision commonly employed. Temporary drainage in this locality gives results otherwise unattainable and the scarring is negligible.

When anatomic anomaly in the shape of a deep pocket in the extreme lateral position precludes the possibility of a cure of this part—and consequently all—of the frontal sinus by drainage alone, the outer part of the orbital ridge is removed through the external or supplementary incision. The external part of the frontal sinus is thus obliterated. This does not cause much disfiguration. It is a thoroughly practical and valuable modification.

The lining of the frontal sinus should be removed as completely as possible if diseased.

Treatment of the Ethmoid Sinuses.—The ethmoid cells are removed carefully under direct vision. Those extending laterally are reached and obliterated by removal of the orbital roof where it forms the floor of the cells. These cells are removed cleanly up to the roof or dural plate and back to the sphenoid. The mucosa of the nose and that covering the medial wall of the ethmoid mass is carefully guarded while the underlying bone, including the superior and middle turbinates, is removed. This mucosa is used in forming flaps, as will be described.

Treatment of the Sphenoid Sinus.—The sphenoid sinus is entered through its thin anterior wall. The condition of the lining mucosa is carefully studied. If the mucosa appears normal it is not disturbed. The anterior wall or sphenoturbinate is removed, care being taken not to injure the sphenopalatine artery.

If, on inspection, the sphenoid mucosa is obviously diseased or the clinical symptoms demand its removal, a difficult problem presents itself.

The literature of the pre-ligation period is filled with evidence of the trouble that was encountered in the surgical treatment of the

sphenoid sinus when the lining was diseased. Much stress was laid upon the difficulty encountered when that lining was curetted or removed. Granulation tissue, polypoid, purulent masses defeated every effort. We read warnings against tampering with the diseased mucosa because of the repeated occurrence of purulent polyposis or the formation of purulent crusts. It is written that crusts are the necessary consequence of too much nasal space.

In refutation of this fallacy the lesson might have been learned from common experience in general surgery. An incompletely drained abscess elsewhere in the body gives rise frequently to a fistulous tract lined by exuberant, polypoid granulation tissue. Neither polyposis nor crusting occur in a nose where drainage is correctly established.

The sphenoid sinus is often a large obliquely placed cavity. Removal of the thin anterior wall or sphenoturbinal did not suffice to afford drainage. Attempts to remove the floor necessarily entailed damage to the sphenopalatine artery. Hemorrhage from that large artery stopped further operative nicety. Further progress in the treatment of the sphenoid sinus demanded, first, control of the sphenopalatine artery.

The method developed for the ligation of the sphenopalatine artery met this problem. It has stood the test of time. Operating, in 1926, before a number of my peers, I was embarrassed by inadvertently cutting the sphenopalatine artery. A dry field was immediately flooded. A previously quiet patient began to become unruly. Fortunately the vessel was ligated. The work then proceeded smoothly and assurance was given that the artery could be successfully handled.

When, after due study, one has decided to obliterate the sphenoid sinus the procedure is as follows: The thin anterior wall of the sphenoid has been removed. One can look directly into the sphenoid at the bottom of the operative field. With a long sharp periosteal elevator the mucosa is raised from the nasopharyngeal wall under the floor of the sphenoid. This periosteum containing the sphenopalatine artery is separated from the bone for about 1 cm. The bone which has been denuded represents the anterior part of the sphenoid floor. This is taken away with forceps of strong design. A special needle, threaded at the point, is passed through the mucosa into the sphenoid cavity. The vessel is incuded in the knot that is tied by special ring "tyer" already used in tying the ethmoid vessels.

Instead of proceeding at once to the removal of the floor of the sphenoid an involved procedure has been found by experience to be required. Many sphenoids are 3 to 4, even 5 cm. deep, while the distance between the rostrum of the vomer and the lateral wall—the width of approach—is not, in some cases, more than one centimeter. It is physically impossible to cut out bone, often heavy, for that great distance through such a narrow approach. Also, if technically possible to cut such a narrow ditch, granulation tissue would soon grow across and obliterate it. This disappointing experience led me to develop the following technic.

If the floor is to be taken away, it is obvious that greater width must be found for the approach. This necessary space may be gained by utilizing the other sphenoid. A submucous resection of the nasal septum is performed. If there is any septal deformity, the incision is made anteriorly far enough to include it. If there is no septal irregularity of importance, what I call "a posterior submucous resection" is done. The incision through the septal mucosa in that case is made over the posterior part of the vertical plate of the ethmoid and vomer. The thin vertical ethmoid plate is easily penetrated by even a blunt elevator. The septal mucosa is dissected free from both sides of the septum. It is spread apart and removed from the front of both sphenoids. One sphenoid has already been entered, the other is now opened from between the layers of the septal mucosa. The back part of the bony septum is removed with strong forceps. Next the rostrum of the vomer and then the sphenoid septum are removed. Both sphenoids have now been converted into one cavity. The approach is limited on either side only by the bases of the pterygoid processes. Working through the facial incision, the flaccid septum is pushed aside and the mucosa of the fornix of the nasopharynx is elevated from the sphenoid bone as far as this makes the floor of the sphenoid sinus. There is now ample room to remove the entire bony floor of both sphenoids. No floor whatever should be left to interfere with perfect downward drainage if obstinate cases are to be cured. After the bony floor has been removed, or while doing so, the mucosa is cut away underneath on the side on which the sphenopalatine has been tied. Experience has proven that it is not necessary to tie the opposite sphenopalatine artery nor resect the nasopharyngeal mucosa on that side. The drainage from that sphenoid is down over the intact mucosa.

My own experience in this connection has been illuminating. Impelled by the same humanitarian motive that led the kind gentleman to cut off his dog's tail an inch at a time, in a certain case, it

took me four attempts to remove a very thick sphenoid floor. Until all the floor was removed there was recurrence of a purulent granulating mass. The floor once completely removed, the mucosa healed smooth and clean without crusts.

The fronto-ethmo-sphenoid operation as here briefly outlined is admittedly subject to fair adverse criticism. We can state in its favor that it represents a more thorough practical technic than has heretofore been presented. We can also claim a much greater percentage of so-called cures than by any other method. It has taken advantage of technic advanced gradually through the years. To its discredit we must admit that it does not always cure. Most of the trouble comes from the frontal sinus. The ethmoids and the sphenoids are obliterated by this technic. The frontal sinus is not obliterated. Seldom is the general surgeon asked to cure an abscess without obliterating the abscess cavity. The tuberculous abscess in the lung and the pleural empyema have both at last been met by the surgical principle recognizing the necessity for the obliteration of the abscess cavity. The dentist at last has abandoned the abscessed tooth. The rhinologic surgeon, however, is asked to handle the abscessed frontal and maxillary sinuses without obliteration of their cavities. Our only chance for cure, therefore, depends upon adequate and permanent dependent drainage, not simply "aeration." Satisfactory drainage downward through the exenterated ethmoid mass may be expected immediately following the technic outlined. Ordinarily this opening can be counted upon to satisfactorily combat the inflammatory conditions accompanying an acute sinusitis. A weakness, however, inherent in the principle employed, allows the wide frontonasal opening, left after operation, gradually to narrow and even close completely in a small percentage of cases. The removal of the floor of the frontal sinus, the frontal process of the maxillary bone and the lamina papyraceæ take the support from the soft parts overlying. They are gradually drawn inward by granulation and scar tissue until the immediately satisfactory postoperative frontonasal opening may be narrowed or completely closed.

Much thought and effort have been spent upon the problem of keeping this nasofrontal space open. The removal of the bony window is essential to entering the operative field. This includes the anterior part of the lamina papyracea. As this part of the lamina cannot be preserved, the rest of the lamina is perhaps best sacrificed. It makes the technic easier, allowing one to follow the ethmoid cells more easily toward the orbital apex. Also it eventually allows the orbital soft tissues to crowd over and encroach on the exenterated

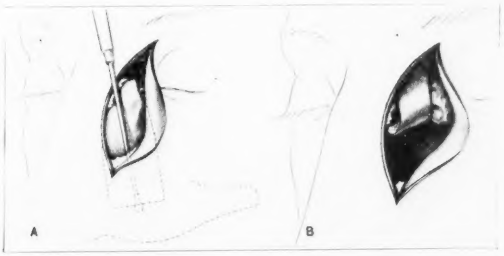


Fig. 1. *A*, the nasal mucosa has been bared. The separation of the flap and the approximate extent of flap are shown. *B*, the flap of nasal mucosa attached above is turned upward to line the frontonasal opening and the sinus.

ethmoid region. It thus reduces the nasal space advantageously. Many methods have been employed for keeping the frontonasal opening patulous. The long wearing of a rubber tube, dilatation by sounds, skin and mucous membrane grafts, have all been used with varying success and failure. When the frontonasal opening closes, drainage from the frontal sinus stops. Unless the diseased cavity has become obliterated by new formed tissue trouble is to follow.

To prevent the disastrous closure of the frontonasal opening I have developed a flap. This is made of the nasal mucosa, principally from the agger region of the nose.

The Frontonasal Flap.—The frontonasal flap is formed and disposed as follows: Working through the facial incision and bony window, the nasal mucosa, which has been carefully preserved, is separated from the nasal process of the maxillary bone. This is accomplished by passing variously curved periosteal elevators downward. The mucosa can be elevated well down, even off the medial surface of the inferior turbinate. Working from the anterior naris, a horizontal incision through the nasal mucosa down upon the underlying elevator outlines the lower free border of the flap. Vertical incisions accomplished through the naris and through the external incision complete the formation of a quadrilateral flap, free below and attached at the roof of the nasal fissure. This sturdy piece of mucosa from 1 to 3 cm. in width and from 3 to 6 cm. in length is turned upward to line the medial wall of the frontonasal opening and more or less of the frontal sinus. After months of experimentation with this flap and the ethmoid flap to be described I was hesitant, working on the cadaver, because of the friability of the embalmed mucosa. Fortunately the interest of the Los Angeles study group, before whom

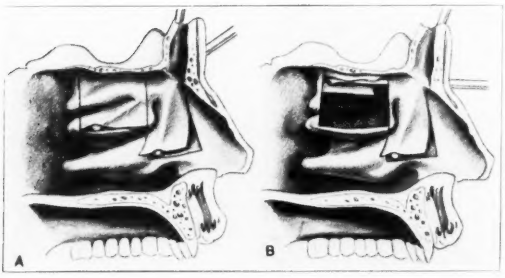


Fig. 2. *A*, the preparation of the two flaps is shown from the nasal aspect. *B*, the ethmoid flap is being smoothed over the denuded surface of the ethmoid roof. The frontal flap is still represented in the nose.

I operated in January, 1934, induced me to try these modifications on the living. I found the mucosa tough enough for the necessary manipulations and the technic practical. I now have been using the flap for a year and am much encouraged by the fact that none of the openings into the frontal sinus have closed. It puts the operation for me on an entirely new footing.

The Ethmoid Flap.—The ethmoid mucosa covering the medial aspect of the ethmoid mass is now disposed of as follows: All the thin bone, including the medial plate of the superior and middle turbinates, is carefully removed from this mucosa. This sheet of mucosa is plastered over the ceiling of the ethmoid mass, i. e., the inferior surface of the dural plate covering the lateral mass of the ethmoids. The median bony wall of the ethmoid mass has been trimmed down almost level with the cribriform plate. This can be done with perfect safety with sharp-cutting bone forceps, working with perfect vision in a bloodless field. The mucosal flap covers the denuded bone from the cribriform plate laterally.

A flap similar to this was described by Monson in the Archives of Otolaryngology for January, 1934. This is ingeniously conceived by the author for use with the intranasal technic. My ethmoid flap for use with the external technic was developed after a long period of experimentation and applied before this article reached me. I believe the principle will find its greater field of usefulness in association with the external operative approach. The necessarily incomplete nature of the work done by intranasal ethmoid technic has been too well recognized to need emphasis. In view of that fact one might fear the sealing of partly exenterated ethmoid spaces.

STANFORD UNIVERSITY HOSPITAL.

XXXII.

AN INTRAMUCOSAL TEST FOR HYPERSENSITIVITY IN ALLERGIC RHINITIS.*

L. W. Dean, M. D.,

Lloyd D. Linton, B. S., and

C. S. Linton, M. D.,

St. Louis.

Marked differences in the structure and physiology of the mucous membranes as compared with the skin would lead one to expect differences in their reaction to irritants. This is borne out by the observations of Duncan[1] and of Cooper,[2] who showed that poison ivy leaf (Rhus toxicodendron) may be chewed with impunity by susceptible persons, but contact of the leaf or saliva with the skin about the month results in a severe reaction. The nasal mucosa is the primary shock tissue in cases of allergic rhinitis. Nasal tests, therefore, should be more specific and give more reliable information than skin tests in such cases.

The advantages of the skin tests are principally the relative ease of application, the large number of tests that can be run at one time, and the relative freedom from severe reactions. The failure of the skin to react in many cases of allergic rhinitis, and its tendency to react to great numbers of allergens that the nasal mucosa is not sensitive to, reduces the value of the test as a diagnostic procedure in this condition. Rackemann and Simon[3] found 8 per cent of positive skin reactions in persons showing no symptoms of hypersensitiveness. It is obvious that any of the various methods of parenteral administration of antigenic substance, which results in absorption into the blood stream, may cause sensitization of the skin.

The limited number of tests that can be performed, and the severe symptoms often resulting from the methods of testing in vogue, has prevented general use of mucosal tests in the nose.

The first recorded test for hypersensitiveness is a mucosal test done by Kirkman[4] on himself in 1835. He sniffed pollen from sweet

*From the Department of Otolaryngology, Medical School, Washington University. This investigation was made possible by the Ball Research Fund.

vernal grass and induced a severe attack of hay fever. Blackley[5] (1873) tested various pollens by conjunctival, nasal and buccal instillation and carried out the first systematic study of pollen hypersensitiveness. Dunbar[6] (1903) placed chief reliance upon the conjunctival test, but used the intranasal method also. The skin test did not come into use until 1907. Some recent reports have appeared on the use of mucosal tests. Efron and Penfound[7] blowed pollen into the nose with a blower and secured positive reactions in eighteen out of twenty fall hay fever cases. The symptoms were those of hay fever with varying degrees of severity. The onset of symptoms varied from a few minutes to fourteen hours and lasted ten days in one case and fourteen days in another. They conclude that cutaneous reactivity bears no constant or quantitative relationship to the clinical picture, and that the nasal test is the most physiologic method, as well as the more practical, more specific and more convincing. Rudolph and Cohen[8] applied a cotton pledget slightly moist with allergen to the nasal mucosa in cases of vasomotor rhinitis with negative skin reactions. He secured positive reactions in nineteen out of twenty-two cases. One patient had pruritis and other symptoms for a week following the test. Stevens[9] exposed the tracheobronchial epithelium in forty-five asthmatics by having them inhale a fine spray through the mouth. Eleven skin negative cases were also negative by inhalation. Fifteen of the remaining thirty-four cases reacted to pulmonary tests and about one-half of the fifteen showed weak or negative skin reactions. Some of the more sensitive cases had asthma lasting for several days.

Intramucosal injections or mucosal scratch tests have not been described, so far as we have been able to determine. This is a preliminary report of the method of using these two tests in making a study of allergic rhinitis.

Technic of Mucosal Tests.—Intramucosal test: It was necessary to have made a needle which could be used conveniently in the nose and at the same time be so small that the puncture of the turbinate could be made by direct inspection with reflected light. Some of the earlier tests were performed with an ordinary bent, gold dental needle. It was fitted to a Luer syringe with an adapter. However, the needle was too large and leakage occurred between the adapter and needle after one sterilization. A special gold needle made on the same general plan but smaller was then devised. It has a 30 degree angle about 15 mm. from the end with a point of 26 gauge and 7 mm. long. The needle fits directly on a 1 cc. tuberculin type Luer syringe. It is desirable but not necessary to have a thumb ring clipped

on the end of the syringe plunger, and a spool on the barrel, in order to steady the grip in making injections. A little practice will enable one to make the complete intramucosal injection with one hand under direct vision.

The tests are performed between the periods of acute allergic reaction. The surface to be tested, usually the anterior end of the inferior turbinate, is swabbed with 1 per cent cocain solution a few minutes before injecting to reduce sensitivity. One test substance is injected on each side. The syringe is loaded with the proper quantity and concentration of test substance (.01 cc.) for an injection, the needle is introduced into the nose through a nasal speculum, and the injection made as shallow as possible in the mucosa a short distance back of the tip of the inferior turbinate. A small pledget of cotton is placed over the area for one or two minutes to catch the drop of blood which usually exudes. It is difficult to make the injection so there is no trace of bleeding. The patient is kept under observation for thirty minutes to one hour and then instructed to observe symptoms to be reported at the next visit.

Mucosal scratch test: The technic of this test is similar to the skin scratch test, using the same needle and syringe as for the intramucosal test. The region to be tested is swabbed with 1 per cent cocain, and the empty syringe and needle used to make two short scratches on the inferior turbinate or inferior border of the middle turbinate. Some of the allergens on a small, sterile cotton swab is then rubbed over the scratched surface. It is convenient to remove the allergen from the bottle with the sterile needle and syringe and squirt it onto the cotton swab. The same outfit may then be used for making the scratches.

Diagnostic Use of Mucosal Tests.—At least one injection or scratch control using saline should be made on each patient undergoing a series of tests. However, there are usually some negative reactions in any series which are sufficient controls on the effect of trauma. The reaction of two substances may be tested simultaneously by these methods, one on each side of the nose. The local reaction in the nose is sufficient to indicate whether one or both substances have reacted. Should there be any doubt as to the etiologic relationship of a test substance to the reaction, each one may be tested separately at a later date. It was found impossible to do more than one test at a time on each side of the nose and tell which substance was giving the reaction. Consequently nasal mucosal tests require many more days than skin testing. The reactons vary with the patient, but a positive

reaction is usually indicated by some local edema and swelling of the turbinate in fifteen to thirty minutes. A pitted appearance of the turbinate was present in some instances when the reaction was fully developed. At times a perfect wheal is produced on the turbinate. Later symptoms consisted of nasal blockage, increased nasal secretion, which may simulate nasal hydrorrhea, sneezing, headache, asthma or other symptoms characteristic of the case. Some reactions are delayed for several hours so that no more than two tests can safely be performed within twenty-four hours. Symptoms from a positive reaction should subside before further tests are made.

The onset of symptoms varied from five minutes to eleven hours with all but four beginning within three hours and a majority of them within one hour. There were no severe reactions. One case had violent sneezing, which was readily controlled by ephedrin. In another case it was necessary to use adrenalin packs in the nose. The duration of symptoms varied from a few minutes to four days. Most cases were completely recovered within five hours. One patient had nasal blockage, which had not been present before, lasting for four days, and another had nasal discharge and blockage for two days.

All cases in this series had preliminary skin tests and in most cases passive transfer of the blood serum had been done. When a strongly positive skin test was secured a preminary mucosal scratch test was done or a weak extract of the allergen injected. This procedure may account for the lack of annoying reactions. Substances such as horse serum and ragweed, to which high degrees of sensitivity may be developed, should always be used with great caution. The history may give some hint as to what may be expected. The fixation of antigen in an area of local sensitization may make the danger in this method of testing more apparent than real when used in cases sensitized to inhalants.

The intramucosal test has proven the best method for securing information as to the sensitizing proteins in allergic rhinitis and the virulence of their action. Furthermore, exact information is secured in this way which may be used in determining the result secured by treatment. It has been most helpful in our investigative work. The fact that a known quantity of the antigen is used is a distinct advantage in this respect. Previous methods of testing by inhalation, powder blowers, sprays, etc., are very inexact and a critical dosage with severe reaction is likely to result. A comprehensive group of staff members of the Department of Otolaryngology is making a study of the various treatments that favorably influence allergic rhinitis. Com-

bined with the clinical study is a very careful nutritional, chemical, immunological and physiological investigation. A cytological study of secretions, blood and tissue is being made. In the group is an immunologist, a bacteriologist, an expert in nutrition, one in tissue culture, a cytologist, a chemist, a physiologist and a physicist. This test, together with a study of skin sensitivity and passive transfer with blood serum, has been made on many of the research cases before and after ionization. This group hopes soon to be able to report the results of the work, particularly the indications for and contraindications for the use of ionization in cases of allergic rhinitis.

It is not possible to draw conclusions as to the comparative diagnostic value of skin tests and mucosal tests from the limited amount of data available. However, there are some observations that are of interest. Case E. W. was found to have all negative skin tests. The nasal mucosa reacted to milk, and on repetition the skin test for milk was found positive. The mucosal and skin reactions were the same in many instances. Four cases showed positive mucosal with negative skin reactions for one to three substances. Two of these four cases were definitely nonreactors to all intradermal tests, while the other two reacted slightly to only a few substances. One case showed a positive intradermal test to and negative mucosal test to milk and eggs.

The use of mucosal tests may be nicely illustrated by the following case: A. B. S. had a diagnosis of allergic rhinitis and sinusitis without infection. There were myriads of eosinophils in his nasal discharge. He had an 8 per cent blood eosinophilia. There was the usual great thickening of the lining of the nasal sinuses with the serum logged mucous membrane of the nose, with a slight swelling of the turbinates, which were paler than normal. It was very important to find the offending allergens in this case. The skin tests were negative with the exception of a plus-minus (doubtful) reaction to banana. The mucosal tests gave the following reaction: Whole wheat was injected; a 1/100 cubic centimeter of a 1/10 dilution, on the anterior end of the inferior turbinate. Following this there was blockage of the right nasal passage in three hours with swelling of the turbinates, severe headache on the right side and a sore throat or pharyngitis. These symptoms lasted eight hours.

Mucosal Tests Before and After Ionization.—Some interesting, if inconclusive, results have been secured by doing mucosal tests before and after the ionization treatment for allergic rhinitis. One case (L. G. P.) showed a good mucosal scratch reaction to ragweed

TABLE 1.

Patient	*Diagnosis*	*Treatment*	*Blood Eosin*	*Skin Tests* +	*Passive Transfer Bloom Serum* +	*Mucosal Tests* −	*Mucosal Tests* +	*Reaction to Mucosal Tests*
E. W. 25 yrs. white	Allergic rhinitis	Ionization	5%	Milk 1+ Others negative		House dust	Milk Milk (M. turbinate)	Itching, sneezing, blockage after 8 hrs. and lasting 12-18 hrs. (In allergic looking area) Reactions same with less swelling.
I. R. 23 yrs. white	Allergic rhinitis	Nutritional	16% & 5%	Eggs+ Potatoes+ Plum± Rye± Asparagus±	Neg.		Beef Potato± Egg±	Slight edema 15 min. Blockage and sneezing sensation in 2 hrs.
F. B. 26 yrs. white	Allergic rhinitis	Ionization	7% & 9%	Horse dander Ragweed Orris root Black pepper Pyrethrum Milk Eggs Chocolate Mixed nuts	Chocolate Ragweed Mixed nuts	Egg Milk	Chocolate Horse dander Ragweed Orris root Pyrethrum Apple House dust	Blockage, sneezing, discharge beginning 2 min to 5 hrs. 8 mal. edematous area early. (Some with all allergens.)
W. S. 50 yrs. white	Allergic rhinitis	Submucous Polyps Caldwell-Luc	3%	Banana Ragweed White flour Cottonseed Several±	Ragweed White flour Cottonseed Banana		White flour } Ragweed } Cottons'd 2+ } Ragweed 4+ }	Symptoms head cold in 10 min. Swelling of turbinates. Nasal discharge. Nasal blockage for 4 days.
H. W. 45 yrs. white	Hay fever Asthma	Ionization	2%	Ragweed Many±	Ragweed Chocolate		Ragweed	Headache and symptoms "severe cold" lasting more than 24 hrs.
W. R. 26 yrs. col.	Allergic rhinitis Polyps Defl. septum	Submucous	3%	All neg.		Goose feathers Chocolate Tomatoes Wool Ragweed Egg Pork Potatoes Tobacco	House dust Milk White flour	Blockage, itching and burning beginning in 3 hrs. and lasting 30 min. Blockage only in 20 min. and lasting 1 hr.

E. F. 20 yrs white	Allergic rhinitis	Adenoidectomy Diathermy turbinates Cautery turbinates Ionized Rt.	4%	All neg.		House dust Chocolate Milk	Goose feathers Timothy Oats±	Nasal blockage and sneezing sensation in few minutes and lasting 2 days. Slight edema 20 min. Nasal blockage in 2 hrs., lasting 2 days. Plain in region eyes.
O. K. 39 yrs. white	Hay fever Asthma	Ionized	3%	Fish Milk Goose feathers Duck feathers Chicken feathers Orris root House dust June grass Timothy Pyrethrum Pea Mixed nuts Wool Ragweed 1:100,000 Many ±	Orris root Horse dander Milk Ragweed Mixed Nuts Goose feathers	White flour St. dust	Fish Ragweed 1:100,000 Milk Goose feathers Ragweed 1:1000 1 mo. after Ionization Ragweed 1000 Fish 1-10	Slight local edema from ragweed in 20 min. Mild asthmatic attack in 5 hrs. Slight local edema from milk in 20 min. Sneezing sensation and asthmatic attack. Slight blanching 20 min. with drawing sensation several hours. Increased nasal discharge in 5 min. with sneezing and swelling of the turbinate. Passage completely blocked in 15 min. with slight headache and slight asthmatic attack. Adrenalin pack applied Turbinate slightly swollen in 30 min.
E. M. 30 yrs. white	Asthma Hay fever	Ionization	5-8%	Ragweed ± Mixed nuts ± Others neg.	Wool ± Ragweed ± Feathers ± Tobacco ±	After 2 wks.	Ragweed Wool Goose feathers Mixed nuts { Ragweed { Feathers	Nasal blockage in 3 hrs. and lasting ½ hr. Nasal blockage in 2 hrs. lasting 1 hr. Blocked again ½ hr. later with headache and pain in eyes lasting 24 hrs. Nasal blockage in 2 hrs. with symptoms of cold and frontal headache for 24 hrs. Slight headache and nasal blockage in 2 hrs., lasting 1 hr. Slight headache in 30 min. Sneezing with slight nasal discharge 2½ hrs.
M. C. 30 yrs. white	Allergic rhinitis	Submucous	8%	Chicken feathers House dust Ragweed Banana	All neg.		House dust Chicken feathers Ragweed Banana	Slight edema in 15 min. Itching, sneezing, increased discharge 45 min. Same as house dust. Lasted 5 hrs. Itching and small edematous areas on turbinates in 10 min.—2 hrs. Then headache with sneezing several hrs. Itching with no edema 15 min.—4 hrs. Then headache and sneezing in 2½ hrs. with incr. nasal discharge.

TABLE I (Continued).

Patient	*Diagnosis*	*Treatment*	*Blood Eosin*	*Skin Tests* +	+ *Passive Transfer Bloom Serum*	*Mucosal Tests* —	*Mucosal Tests* +	*Reaction to Mucosal Tests*
S. K. 42 yrs. white	Allergic? Migraine	None Ionization	5%	Orris root Horse dander Pyrethrum	Horse dander ± House dust ±	*Horse dander *Pyrethrum Orris root After 2 wks.	Orris root Orris root	Sneezing and nasal blockage in 30 min. Sl. local edema and sl. blocking in 30 min. No blocking by inspection. No reaction next day to orris root.
H. S. 54 yrs. white	Hay fever Asthma Arthritis	Ionized	1%	Ragweed White flour Rice flour Several ±	All neg.	White flour Ragweed (Scratch)		
L. G. P. 44 yrs. white	Allergic rhinitis	Ionized	11%	Ragweed House dust ± Horse dander ± Chocolate ±	Ragweed ± Feathers ± Chocolate ±	Ragweed	Ragweed (Scratch) House dust (Scratch)	Sneezing, nasal discharge and blocking in 15 min. Pitting rt. turbinate.
I. S. 32 yrs. white	Allergic rhinitis		6%	Orris root Mixed nuts Turnips Pyrethrum Milk House dust Cabbage Rice flour Chocolate Bl. pepper White flour	Orris root		Orris root (Scratch) Milk White flour Mix. nuts Turnips Whole wheat	Blockage, increased discharge, headache. Blockage (unusual), numbness left side head and pain in ears lasting 24 hrs. Nasal blockage. Soreness, local edema, blockage, nasal discharge 75 min., clearing 1½ hr. Slight blocking. Complete blockage.
E. M. P.	Allergic rhinitis		8%	Chocolate Kapok Pyrethrum	Chocolate Pyrethrum Wool ±		Pyrethrum	.01 cc. injected. Slight burning in 10 min.
A. B. S.	Allergic rhinitis	Nutritional	8%	Banana ±		Orange House dust Feathers	Whole wheat	.01 cc. injected. Blockage of right nasal passage in 3 hrs. with soreness of turbinate, fever, headache on rt. side of head with pharyngitis. Symptoms lasted 8 hrs.

NOTE: *Scratch method; all others .01 cc. injected.

before ionization and fourteen days after ionization there was no reaction whatever to ragweed. The blood transfer test was plus-minus (doubtful). Case S. K. gave a good intramucosal and intradermal reaction to orris root before ionization. The mucosal test was repeated two weeks later and found to be still positive. The skin and mucosal test were done again, nineteen days after ionization, and both were negative. This patient had no reagins in the blood. E. M. was positive to ragweed, goose feathers and mixed nuts; two weeks after ionization this patient remained sensitive to goose feathers and ragweed but failed to react to mixed nuts. The blood transfer test was plus-minus (doubtful) to the three substances. O. K. was ionized on the left side, using a zinc electrode and zinc sulphate electrolyte. A strong positive reaction was secured to ragweed before treatment and one month after treatment. On the right side ionization was done with the technic of Alden with the same result. This patient had reagins in the blood. The patient had a good clinical result at this time on each side of the nose. The mucosal test was the same on each side. Alexander and Alexander[10] reported that the presence of reagins in the blood stream suggested a poor result following ionization for hay fever; that reagins had been found absent in most cases having a good result from ionization.

In the perennial type of allergic rhinitis the condition of the mucous membrane of the nose seems to be an important prognostic factor. If we have the nose blocked by a diffuse serum logged membrane with the turbinates so large and boggy that they are flat against the septum, ionization will give an improvement for an indefinite period of time. The question always is: Is ionization the best procedure to use, not will ionization cause improvement? The department is making a serious effort to clarify this subject. It is hoped that by August we can express a definite opinion as to the indications and contraindications for the use of ionization in allergic rhinitis. A group composed of a clinician, a pathologist, a bacteriologist, an immunologist, a cytologist, a physiologist, a physicist and a nutritional expert is working on the problem.

Operative procedures on the nose in two cases had no effect on the mucosal reactions. A Caldwell-Luc was done on one. He remained sensitive to ragweed and white flour twenty days after the operation. A submucous resection on the other failed to alter the mucosal sensitivity to ragweed, house dust, feathers and banana. Both cases, however, were relieved by the surgical procedure.

Miss Zelma Alvis contributed much to these observations by making the skin tests.

SUMMARY.

1. A description is given of a method of performing intramucosal and mucosal scratch tests in the nose.

2. No severe reactions were procured. In fact, one case secured a more alarmng reaction from 1:10,000 intradermal ragweed than from 1:1000 intramucosal ragweed. The skin injection resulted in a lump the size of a hen's egg within thirty minutes, while the mucosal injection produced only marked nasal blockage, nasal discharge and sneezing lasting about four hours.

3. The intramucosal and mucosal scratch tests have not, in the limited series available, produced the annoying reactions reported from the methods of mucosal testing previously in use.

4. Disagreement in the results of skin tests and mucosal tests, in this series of allergic rhinitis cases, has been due in most instances to a reaction from the mucosal test where the skin test was negative.

BIBLIOGRAPHY.

1. Duncan, Charles H.: Autotherapy in Ivy Poisoning. N. Y. Med. J., 104: 901, 1916.

2. Cooper, H. M.: Immunization for Rhus Toxicodendron Poisoning. Med. J. and Rec., 136:297, 1932.

3. Ruckemann, F. M., and Simon, F. G.: Technic of Intracutaneous Tests and Results of Routine Tests in Normal Persons. J. Allergy, 6:184, 1935.

4. Kirkman, W. P.: "Der typische Frühsommer Katarrh." From P. Phoebus, p. 137, 1862.

5. Blackley, C. H.: Experimental Researches on the Causes and Nature of Catarrhus Aestivus. London, 1873.

6. Dunbar, W. P.: Aetiologic und spezifische Therapie des Henfiebers. Munchen. Med. Wchnschr., 50(1):924, 1903.

7. Efron, B. G., and Penfound, W. T.: A Nasal Test with Dry Pollen in the Diagnosis of Seasonal Hay Fever. J. Allergy, 2:43, 1930-31.

8. Rudolph, J. A., and Cohen, M. B.: Vasomotor Rhinitis with Negative Skin Tests: A Local Nasal Allergy. J. Allergy, 5:476, 1934.

9. Stevens, F.: A Comparison of Pulmonary and Dermal Sensitivity to Inhaled Substances. J. Allergy, 5:285, 1934.

10. Alexander and Alexander: Ionization of the Nasal Mucosa and Relationship Between Reagins in the Blood and Effect of Treatment. J. Allergy, 6:240, March, 1935.

XXXIII.

THE DEAFNESS OF BEETHOVEN.*

IRA FRANK, M. D.,

CHICAGO.

The deafness of Beethoven should be of great interest to all lovers of fine music, to those interested in biographic literature and certainly to all otologists. No apology therefore is necessary; indeed, it seems appropriate to consider this subject with a group of oto-laryngologists. Although there is a fair amount of literature dealing with the life and work of Beethoven, it deals mostly with a critical analysis of his musical creations and an appraisal of his influence on the world of music. The books and articles which discuss his deafness are not numerous and are written mostly in the German, French and Italian languages; there is little available literature in English about his deafness. The principal sources to which I am indebted for such information as is given here are mentioned in the appended bibliography.

Although my paper is intended to deal mainly with Beethoven's deafness, it is difficult to refrain from a consideration of the purely sentimental and human aspects of the subject. Who can picture the soul anguish of this most highly gifted and rare genius, whose musical creations have delighted and will continue to delight generations yet unborn, when at the flower of his full creative power he found himself threatened with the loss of hearing which obviously was most vitally associated with his musical creations. Poor Beethoven! How he must have suffered as his diminishing hearing interfered with his artistic activities. He consulted one physician after another for relief from his increasing infirmities, but he obtained no help. What can be more deplorable than the loss of hearing to a musician; or the loss of sight to a talented artist?

We must stand reverently before this prince of musical geniuses. We cannot fail to admire his stoical courage, his indomitable perseverance, his incessant work, his intense love of music, and his unexcelled ability to express through music what he himself felt. Through his beloved art he expressed his own emotions, thoughts and

*Presented before the meeting of the Chicago Laryngological and Otological Society, April 1, 1935.

ideals. Beethoven was not alone a great musical genius; he was also a great man.

Ludwig van Beethoven was born at Bonn, Germany, December 16, 1770. His ancestors lived in a Belgian village near Louvain, from which place some of the Beethoven family migrated to Bonn in the seventeenth century. His father, Johann van Beethoven, was a shiftless man addicted to alcoholic excesses. This habit increased with his advancing age and he died apparently as a result of this addiction. Both Johann and his father before him were professional singers in the Elector's choir at Bonn. Beethoven's mother and at least one brother were tuberculous and died of the disease. The genealogy of the family was worked out in detail by Bilancioni. There does not appear to be any history of internal or middle ear disease on either the maternal or paternal side. Beethoven's musical talent was displayed from his earliest years. He was considered a musical prodigy at the age of four, and at the age of nine he could play well the sonatas of Mozart and Haydn. In his very early years his father gave him such musical instruction as he could, but later young Beethoven studied with other teachers.

The alcoholic and shiftless habits of his father caused much poverty and misfortune, and young Beethoven's life was full of misery and fatigue; he was probably insufficiently and improperly nourished. This accounted for much of his misanthropy in later life. In 1787, at the age of seventeen, possibly with the assistance of the Elector, he was sent to Vienna to improve his musical education under the direction of the great master, Mozart. Owing to his mother's death in the same year, he was obliged to return to Bonn and assume the responsibility as of the grown son of the family. It seems that in his infancy he suffered from variola, after which he developed a severe attack of enteritis. This happened about the time of his mother's death and continued to annoy him for the rest of his life. He also suffered from attacks of asthma and bronchitis, as well as from nasal catarrh, which became chronic. In 1792 he again went to Vienna and made it his permanent home. Later in life he developed icterus, which was probably a forerunner of a serious cirrhosis to which he finally succumbed. He also suffered from myopia, and in his letters he frequently complained of eye trouble, which was probably due to defective vision.

Let us glance for a moment at Beethoven's physical appearance during his manhood, as sketched by his biographers. Bilancioni states that he had a massive head like the cupola of a temple, a sharply defined mouth, signifying indomitable pride, and tempestu-

ous though vivacious blue eyes. He, like Haydn and Schubert, was of small stature, corpulent, of athletic constitution, with a large face, high prominent forehead, rough black hair, small nose with wide nostrils, projecting lower lip and also a projecting lower jaw.

It is not my intention to deal in any detail with the musical side of Beethoven's life. That has been thoroughly and frequently done. The great misfortune which came to Beethoven at the early age of twenty-six was the loss of his hearing. This deprivation will cause disappointment and discouragement to any man, but to a great composer this loss is irreparable. His general health was also poor, and his intestinal troubles, which had preoccupied him for several years, were now entirely overshadowed by the deafness.

Personally, Beethoven was inclined to be careless about hygienic matters. He was accustomed to go into the woods to compose in all kinds of weather, inclement or otherwise, sitting on damp soil for hours without noticing it, quite absorbed in his musical work. Indoors, he would sit, scantily clothed, in draughts, and when he felt tired from overwork he would dip his head constantly in cold water or suck ice. Physicians of this generation would be less concerned by these indiscretions as causing disease than they were in Beethoven's day. In Thayer's biography of Beethoven it is stated that on a hot day in the summer of 1776 he entered his home in a sweat, opened the windows, undressed even to his underwear, and worked in a draught for a long time. As a consequence of this he is supposed to have contracted a severe attack of enteritis, and coincidentally the biographers think the shock sustained by his exposure affected his hearing and marked the beginning of his deafness. As previously mentioned, he suffered a similar attack in 1787 at the time of his mother's death and he may have had similar attacks in the interval. In the light of our present day knowledge we would say that the enteritis played no part in the causation of the middle ear disease and the affection of the eustachian tube, but his biographers suggest a relationship of the two diseases. According to his friend, Franz von Wegeler, Beethoven himself fixed 1796 as the time of the first onset of his deafness and, according to his letters to Wegeler and others, it continued to progress slowly from this time.

Naturally enough, Beethoven was vastly concerned with this increasing affliction, which to him was more serious than all his other maladies and troubles. From 1801 on he was constantly trying different doctors and not from any caprice, but from real concern, since he did not experience any improvement, but on the contrary he was growing worse. He did not hesitate to criticise his medical

advisers and he expressed complaint and dissatisfaction with their efforts. It is well known that otology at this period was still in its infancy and there was little exact knowledge concerning the pathology, diagnosis and treatment of the middle ear. Dr. Johann Peter Frank of Vienna, still remembered as the great reformer in hygiene and sanitation and the leading Vienna clinician of his period, was naturally consulted by Beethoven, but Frank's treatment of his deafness was unsuccessful. The patient wandered about from one physician to another, just as they do in the present day. He visited Dr. Gerhard Ritter von Vering, Dr. Franz Wegeler, Dr. Johann Peter Schmidt, but none of them gave him any relief. He continued his journey among the elite of the Vienna profession and finally, disgusted with regular medical men, he consulted the quacks. In spite of all his efforts for improvement, by 1805 his deafness had increased so much that he was no longer able to conceal it from his family and friends. He was now compelled to use ear trumpets, which were specially constructed for him by Mälzel, the inventor of the metronome. Beethoven was in such despair that in his famous will, written at Heiligenstadt in 1802, and addressed to his brothers Carl and Johann, he avowed his readiness to end his life. He separated himself from the brilliant society of Vienna, which he loved, became morose, left the city and lived in solitude in the country. He occasionally appeared in public up to 1822. He sometimes conducted the orchestra, though his musicians had difficulty in following him. Beethoven could perceive loud or deep sounds, but failed to appreciate middle tones. This made it almost impossible for his orchestra or the singers to cooperate with him. Up to 1814 he played and conducted fairly well. After this he used hearing devices. After 1820 his public appearances as a musical conductor were failures and were a series of disasters for himself, his audience, and those whom he led. The great tragedy occurred in November, 1822, when he broke down completely while rendering the duet scene in the first act of his only opera, Fidelio, which he attempted to conduct in person. At home, however, he continued to compose and to produce several of his finest symphonies; even the magnificent Ninth Symphony was composed under these trying circumstances. He worked not only by his sense of music, but by means of an audiphone attached to his piano which enabled him to hear to a limited degree. Although his peripheric acoustic organs were gone, he still preserved a peculiar mediative feeling for music which he could think out just as a mathematician solves a problem. A deaf violinist would be unable to play in tune, or a deaf vocalist would have difficulty to sing in tune. A pianist with such an affliction would be unable to strike the correct note

accurately. Beethoven's piano playing in the later years of his life was distressing for that reason. But a composer who becomes deaf after he has been practicing his art for some time does not necessarily lose his ability to compose. Many, though not all, composers never use a piano or any other instrument to assist them in their work. Bach, for instance, used to make fun of such pupils of his who wrote at the harpsichord and he dubbed them "knights of the keyboard." The hearing of music is a psychic perception. Complicated scores are conceived and executed in the intellectual spheres. Writing the notes on paper is a mechanical motor function which originates in the brain. Most of the important works of Smetana, the founder of the Bohemian School, were written after he had suffered the total loss of hearing.

In his latter years, it is said that Beethoven indulged somewhat in intoxicating liquors. Perhaps it is true, perhaps not. If true, it may have aggravated the liver cirrhosis which seems to have been the main cause of his death. At any rate, there seems to be no doubt that his death was caused by the unfavorable progress of his liver and intestinal troubles. Late in 1826, while visiting his relatives in the country, Beethoven was exposed to inclement weather and contracted a cold. While still under the influence of this respiratory infection, he set out to return to Vienna, and on the way was obliged to spend a night in an inn which was unheated, consequently he acquired a pneumonia. He reached Vienna and was attended by Dr. Wawruch for the pneumonia, from which he finally recovered. He continued to suffer from liver disease, which was manifestly hepatic cirrhosis. The liver became atrophic, ascites developed, for which he was frequently tapped. He died March 26, 1827, and an autopsy was performed by Dr. Johann Wagner of the Vienna Pathologic Clinic. In his will, Beethoven had expressed the wish that after his death the cause of his deafness should be investigated by his friend Professor Schmidt, but Schmidt had died in 1809—and Wagner's pathologic examination of Beethoven's hearing apparatus was rather incomplete and did not clear up the cause of the deafness. From the autopsy report, as given by Bilancioni and others, it appears that the cartilage of the ear seemed to be especially large and irregularly formed. The scaphoid fossa (fossa helicis), especially in relation to the concha, was very wide and about one-half deeper than normal. The external auditory canal, especially near the tympanic membrane, was covered with a glistening squamous epidermis. The eustachian tube was much thickened, its mucosa tumefied and somewhat strictured toward the bony part. The cells of the mastoid were enlarged and covered with a heavy mucosa. The arteries and nerves of the

ears were affected, though no details were recorded as to the vascular changes, the acoustic nerve on both sides being thinned and atrophic, the left more than the right.

The opinion has been expressed by some biographers that Beethoven was unskillfully treated medically, both in regard to his general maladies and his ear troubles. As already stated, Beethoven lived at a time when otologic treatment had made but little advance; irrigations and galvanism were the principal methods in vogue for treatment of diseases of the middle ear. Insofar as the treatment of hepatic cirrhosis, from which he suffered and to which he succumbed, it can only be said that the disease is a progressively fatal one and under the best circumstances the treatment is expectant and symptomatic. There was some difference of opinion in regard to the exact nature of his fatal illness. Wawruch called it hepatic cirrhosis, and this is the name given in Frimmel's Beethoven Handbuch. Von Breuning stated that it was "peritoneal inflammation"—Beethoven had suffered from violent colics for the greater part of his life.

From the otologic point of view, naturally the most important question which concerns us is, What was the exact nature of Beethoven's deafness? Was it a labyrinthine affection, an otitis media or an otosclerosis? Several opinions have been expressed by medical men who have written on the subject; some attributed the deafness to catarrh of the eustachian tube, some to otitis media, and others to labyrinthine disease associated with other nervous disorders. Otosclerosis has also been suggested, and the older writers conceived the idea that some hereditary stigma in the size and shape of the external ear was responsible for the hearing defect. And then we must record an alleged cause which no one will take seriously at the present day, that his hearing was weakened because of fatigue and excessive use of his auditory apparatus in his musical practice.

Beethoven's own account of his deafness is sufficiently curious to be worth mentioning. This account was given to the English pianist, Charles Neate, who was on friendly terms with the music master. During a visit to Vienna in 1815, Neate suggested a journey to England with the idea that Beethoven might profitably consult some of the British physicians. In reply to this invitation Beethoven gave the following account of his deafness: "I have received all kinds of medical advice. I shall never be cured. I will tell you how the matter stands. I was at work on an opera once." At this point Neate asked if the work was "Fidelio," the only dramatic composition written by Beethoven. "No, it was not 'Fidelio'," Beethoven replied. "I had to do with a capricious and unpleasant

tenor singer. I had already written for him two arias with the same text, with which he refused to be satisfied."

Beethoven then described a visit from this pestiferous singer. "As he knocked at the door, I sprang from my table in such excitement and rage, and as the man entered the room I fell on the floor, as they do in the theatre. When I got up again I found that I was deaf and I remained so. The doctors say the nerve was injured." This incident, since it did not occur in connection with Beethoven's one opera, "Fidelio," must have taken place, it is generally believed, at the time the master was composing his oratòrio, "The Mount of Olives," which was in 1801. But while Beethoven's hearing had been affected before that year, he was certainly not completely deaf. Moreover, he advanced other causes for his loss of hearing than the traumatic one which he described. In 1814 he declared to his intimate friend, Dr. Aloys Wissenbach—a physician of high repute in Austria—that the deafness followed "a severe typhoid fever."

There is another possible cause of which Beethoven said nothing. This was the syphilis which, although discreetly left unmentioned by the majority of the master's earlier biographers, was referred to by Sir George Grove in his article on Beethoven in the first edition of Grove's Dictionary of Music and Musicians (1879). Ernest Newman, the well-known English music critic, discusses the matter in his book, "The Unconscious Beethoven," and makes it clear that the composer suffered from syphilis, for the treatment of which he received prescriptions (still in existence) which relied on the specific action of mercury. While syphilis may have had a causal bearing on Beethoven's deafness, other causes from symptomatic evidences seem more probable. It is interesting to note in passing that Schubert also suffered from syphilis.

Dr. Marage, a Parisian otologist, offered an interesting theory. He ruled out otosclerosis, as producing a different type of deafness; acute influenza, which if it had been present would have produced otitis media and traces of which would have been revealed at the autopsy; traumatism and the resulting cerebral deafness may also be excluded because the tinnitus had been noticed a long time before the trauma occurred. Marage would also exclude syphilis because he believes the deafness caused by this disease is of more gradual development. In his paper, read before the Acádemie des Sciences, Paris, in 1928, Dr. Marage offered his belief that Beethoven's deafness was the result of a labyrinthitis that may have been produced by two main causes, one of them cerebral, the other intestinal. It is possible that a long-standing membranous colitis might have been a focus for

infection of remote structures. The cerebral aspect, Marage thought was important, and he believed that Beethoven's inner ear and auditory centers, hypersensitive, subjected to intensive labor and overwrought, became congested.

With reference to catarrhal disease of the ear, there is no doubt that Beethoven was a constant sufferer from nasopharyngitis which might have been associated with enteritis that existed since his early youth and which was possibly aggravated by his lifelong unhygienic habits. He had sharp attacks of enteritis in 1787, 1796 and later; in fact, he had chronic enteritis. Klotz-Forest thinks that he developed a catarrh of the eustachian tube which gradually became chronic and towards 1799 developed into an acute otitis media. Buzzings and noises were heard in the middle ear but no pains. Although the left ear was the first that was affected, the right was attacked later, removing any question of possible traumatism. Enteritis or typhoid attacks, like any other intestinal infection, could cause a pharyngeal infection spreading to the eustachian tube and middle ear. But, on the other hand, there is not any record that Beethoven had acute otitic trouble following his early attacks of enteritis. Baratoux and Natier disagree with Klotz-Forest's view that there was an otitis media, and say that all the facts point to the disease known as progressive spongification of the labyrinthine capsule with ankylosis of the stapes. Natier thinks it possible that the onset of the deafness might perhaps be attributed to the general bad state of health and to chronic enteritis acting upon a very fatigued ear.

Deafness of professional musicians from continuous excessive use of the hearing organ appears to be confined exclusively to the labyrinth, the external and middle ear being scarcely ever affected. Such alterations as occur occasionally appear to affect the cochlear segment only, and not the vestibular segment of the labyrinth. Unfortunately, the autopsy description in Beethoven's case is too vague to throw any light on this point. But it has been said that musicians may become deaf from abuse of the hearing organs, and audition is known to be greatly disturbed in cases of labyrinthine sclerosis. According to Schweisheimer, some of Beethoven's biographers mention that he suffered from vertigo which, of course, is a labyrinthine symptom.

Dr. Paul Garnault considered Beethoven's deafness to be of a nervous nature. The labyrinth and acoustic nerves were for thirty years the site of an inflammation causing atrophy of the nerves. Klotz-Forest does not agree with this. In Beethoven's case he thinks it was not a lesion of the internal ear, because up to the last he was

able to perceive vibrations transmitted through the cranial bones, which would have been impossible in case of a lesion of the acoustic nerve. He used a wooden stick for bone conduction when he had become completely deaf, to enable him to hear his piano when he composed, placing one part of the stick on the instrument and the other between his teeth. Dr. P. Bonnier also points to the fact that as Beethoven enjoyed all his musical faculties and his auditory memory, it eliminated the hypothesis of a cortical trouble. But he thinks even though Beethoven perceived sounds through his cranial bones it would not eliminate a lesion of the internal ear. There was apparently no vertigo, no nystagmus; in fact, no symptoms of labyrinthine disorder. Bonnier thinks that Beethoven's malady was not of a peripheral or central nervous nature, but more probably a common tympanolabyrinthine sclerosis.

The question of an hereditary aural stigma in Beethoven has not been dwelt upon at any great length by his biographers. The fact that his wide nasal fossæ might have allowed access to organisms which tended to spread to his ears through the eustachian tube has been pointed out. But Bilancioni mentions the interesting fact that the lobes of Beethoven's ears were not normal in shape; they were flat and the cartilage was deformed. This was referred to in Wagner's autopsy report. Bilancioni gives an illustration of the defective ear lobes. It is rather singular that Mozart's ears were of the same type with an elementary development of the anti-helix and absence of the antitragus. But, even admitting such defects in the external ear, it appears rather difficult to conceive how they could affect Beethoven's hearing apparatus. Following the autopsy, Beethoven's ears were preserved in the Anatomical Museum in Vienna, but when, many years later, Politzer desired to examine them for study, they had disappeared, so we are unable to say whether or not there was any anatomic defect in the middle or internal ear.

Bilancioni, who has written more extensively than anyone else on the question of Beethoven's deafness, and who appears to have studied everything that is known about it, expresses the opinion that the deafness was due to otosclerosis. This aural disease, which received its name of otosclerosis from Politzer in 1893, has no premonitory symptoms. "For me," says Bilancioni, "it is unquestionably a matter of an otosclerosis in the sense of a morbid process clinically characterized by a chronic progressive deafness analogous to the deafness produced by chronic hyperplastic catarrhal otitis media."

Otosclerotic deafness is characterized by qualitative and quantitative disturbances. There is a lesion of the sound transmission as

well as a lesion of the perception. Patients become aware of otosclerosis only when the functional disturbances of the hearing apparatus have reached an advanced stage. The disease generally begins after the advent of puberty, from the sixteenth to the eighteenth year, at first unilateral but soon bilateral. The cause of otosclerosis is today considered to be of a constitutional nature.

All the evidence in Beethoven's case appears to favor Bilancioni's opinion, and with it one is forced, in the absence of more decisive proof of any other condition, to be in agreement. After full consideration of all opinions adduced as to the possible cause of Beethoven's deafness, one is inclined to agree with those who consider the progressive deafness due to an otosclerosis. Unfortunately, the results of the original postmortem are inconclusive, and it is equally unfortunate that the anatomic specimens were lost from the museum. Consequently, definite opinions as to the nature of the lesion are conjectural and the exact nature of Beethoven's deafness will forever remain unsolved.

104 S. Michigan Ave.

REFERENCES.

Baratoux and Natier: La Surdité de Beethoven. Chron. med., Paris, 12:492-496, 1905.

Bilancioni, G.: La sordita di Beethoven: Consideratione di un otologo. Rome, Formiggini, 1921.

Bonnier, P.: La surdité de Beethoven. Chron. med., Paris, 12:521-524, 1905.

v. Frimmel, Th.: Neue Beethoviana, and Beethoven Handbuch (two vols.). Leipzig, Breitkopf, u. Haertel, 1926.

Garnault, P.: Chron. med., Paris, 12:522, 1905.

Jacobsohn, L.: Ludwig van Beethoven's Gehorsleiden. Deut. med. Wchnschr., 26:1282-5, 1910.

Klotz-Forest: La surdité de Beethoven. Chron. med., Paris, 12:321-331, 1905, and 13:209-241, 1906.

Kerst, F.: Beethoven im Eigenen Wort. Berlin, 1909.

Lombroso, D.: La sordita fra i musicisti. Rivist. music. ital., 1:524, 1894.

Prat: De la surdité chez les musiciens. J. d'hygiene, 1:6, 1875.

Rolland, R.: Beethoven: sa vie. Paris, 267-290, 1903.

Schindler, A.: Biographie von Ludwig van Beethoven. Munster, 1840.

Schweisheimer, W.: Beethoven's Krankheiten. Muench. med. Wchnschr., 51: 1437, December 17, 1920.

Thayer, A. W.: Ludwig van Beethoven: Leben. 3 vols., 2:91, 1872. (Translated into English by Krebiehl.)

Wegeler, F. G., and Ries, F.: Biographisches Notizen ueber Ludwig van Beethoven. Koblentz, 1838.

Newman, Ernest: The Unconscious Beethoven. Alfred A. Knoff, New York, 1930.

Sonneck, O. G.: Beethoven: Impressions of Contemporaries. G. Schirmer, Inc., New York.

XXXIV.

INDICATIONS FOR THE BONDY TYPE OF MODIFIED RADICAL MASTOID OPERATION.

H. I. Lillie, M. D.,

Rochester, Minn.

Better results have been obtained in my hands from use of the Bondy type of modified radical mastoid operation in certain selected cases than from use of the typical radical mastoid operation in similar cases.

Körner, in 1899, said that in certain selected cases the radical mastoid operation may be so modified that both the tympanic membrane and the ossicular chain could be kept intact and in position. Heath, in 1904 and in 1907, reported a group of cases in which a modified radical technic was used in mastoid operations. The removal of the tympanic membrane and the ossicles was avoided with the idea of conserving the hearing. In 1906, Bryant described a meato-mastoid operation; his main purpose in not disturbing the middle ear was to conserve the hearing if possible. He described a case of acute exacerbation of a chronic disease in which the operative findings showed that there was a broken down mastoid process.

Kopetzky, in 1909, wrote a critique of these modified mastoid operative methods which were suggested by Heath and Bryant. In this he claimed to have established the following facts: 1. "That the simple mastoid operation, if performed on the same class of cases, would yield as good results in regard to the hearing as does the modified radical operation, and would also better meet the surgical indications in these cases." 2. "That the modified radical mastoid operation does not meet any of the requirements of radical mastoid surgery." 3. "That in cases of otitis media purulenta chronica of long duration the modified radical operation cannot from a scientific standpoint restore to functional activity a diseased (necrotic) ossicular chain or heal necrotic areas in the annulus tympanicus or in the tympanic, adital or antral walls." 4. "That as a substitute for the simple mastoid operation it offers no advantages."

Bondy, in 1910, described a method that more completely fulfilled the surgical requirements. In 1911, Richards compared the

technics of these several authors: Heath had never described his technic, so Richards visited Heath's clinic and saw the operation used and the results obtained. Heath apparently had no definite indications for the operation, because he approached every case with the idea of doing, if necessary, a typical radical mastoid operation. This was done particularly if cholesteatomas were found, and apparently made no difference whether the drums were perforated or not; he also used the operation in subacute cases. Some of the results observed were good, others not so good. Bondy, however, used his method only in chronic cases. He did not regard Shrapnell's membrane as part of the drum. In carrying out his operative technic, he did not disturb the membranous meatus until the final step in the operation, and emphasized the importance of removing the lateral attic wall. Bondy's operative technic differs from the typical radical mastoid technic in that separation of the membranes was done after the bone operation had been completed, and the bridge was made as thin as possible but was not removed. Heath did not remove the lateral antral wall; Bondy said that the drum does not need a bony support. Richards saw several cases in which healing had taken place following employment of this technic. Richards felt, after having seen these operations performed by the men who advocated the technic, that there was a definite field of usefulness for the method in certain selected cases. In otologic literature at this time were many reports of cases in which this type of operative technic had been used. In most of them, however, very indifferent results were reported. In 1916, Blackwell described the results obtained with a modified radical mastoid technic for chronic suppurative otitis media, based on his observations in thirteen cases. His best results were obtained in cases in which there had been discharge from the ears for less than five years. He used the operation in nine cases in which perforations in the drums were present. In six of these cases the drums had healed. Graham felt that there was a certain field of usefulness for the modified radical technic in selected cases.

Unfortunately, in otologic literature there is no satisfactory classification of chronic suppurative otitis media and mastoiditis. The term, "chronic suppurative otitis," as used, is too inclusive; for that reason it is difficult to understand what particular type of case is being reported. All otologists recognize that there are certain types of cases that fall into rather definite groups. At the clinic we have divided on an empirical basis cases of chronic suppurative otitis media and mastoiditis into four classifications; in this classification the present cases (table 1) fall into types 3 and 4.

In my opinion cases that are best suited for the modified radical mastoid operation are those in which there is a history of a chronic discharging ear; examination indicates that there is foul-smelling pus in the canal, and closer inspection reveals that the drum is intact and that there is no evidence of actual middle-ear disease. In searching for the source of pus it is seen that there is a marginal defect into the attic in the region of Shrapnell's membrane, or posteriorly through the bone into the tympanic antrum, and that this defect appears to be too small to permit of adequate, direct local treatment. Roentgenologic examination reveals that the mastoid process, as the result of long standing infection, has become sclerotic and contracted. Usually in this type of case the hearing is good, and it is important that the hearing, if possible, be conserved, although in my opinion this is not the most important feature unless the involved ear is the only hearing ear.

Well directed treatment of the ear should be carried on over a period of time, because even with these unfavorable conditions one is occasionally able to effect a cure by such treatment. If continued observation makes it apparent that local treatment is not going to cause resolution of the disease because of the evidence of necrosis of bone or cholesteatoma, then some operative interference must be contemplated.

Contraindications to the application of the modified radical mastoid operation, in my estimation, would be the presence of perforation or perforations of the drum which to me would mean evidence of middle ear disease. If the mastoid process is shown to be cellular, conditions are also favorable for good results, if the drum is intact. The defect into the attic or tympanic antrum precludes the use of the complete (simple) operation because in all probability the cholesteatomas will reform.

If operation could be accomplished safely by intra-aural procedures and diseased tissue removed, the surgical requirements would be fulfilled. However, it is obvious that adequate exposure of the field by the intra-aural technic would be difficult, might be quite impossible, and obviously would be very dangerous.

Some years ago I observed a patient who was sent to the section on otolaryngology and rhinology for examination because of a history of a chronic suppurative otitis media in childhood. On examination the drum was found to be intact but there was a pear-shaped cavity in the region of the attic and tympanic antrum. No

operation had ever been performed on this ear. It occurred to me that in this particular case a cavity similar to what would have been made by a modified radical mastoid operation had been fashioned by natural reparative processes. I had often wondered if Stacke had not made the same observation when he suggested his operative technic for chronic suppurative otitis media. It would seem that in the type of case just mentioned the modified radical technic of the type suggested by Bondy would better meet the surgical requirements than the typical radical mastoid technic, not because it conserved the hearing but because it did not expose the middle ear. All otologists must have observed that if the membrane on the promontory is secreting normally it is practically impossible to obtain a dry middle ear with the radical mastoid operation. For that reason, then, if the drum and ossicles can be preserved, and the promontory not exposed, the result should be better in this type of case.

I quite agree with Kopetzky in his objections to the Heath technic and the indications for its use, but I believe the Bondy type of operation would meet Kopetzky's requirements because it is used for chronic conditions, thoroughly removes diseased tissue, and the results obtained from it are satisfactory.

The approach I have used in performing this operation differs somewhat from that suggested by Bondy. The first step is a circular incision, made with a right-angled knife, in the membranous meatus, starting just external to the tympanic attachment at the anterior aspect of Shrapnell's membrane and extending around posteriorly to the region about the center point of the membranous meatus on the floor. After incision has been made to the bone, the membranous meatus is partially separated toward the external ear. My idea in using this incision was to avoid tearing the attachments of the drum to insure its integrity. The epitympanum and tympanic antrum were approached after the method of Stacke; in fact, the Stacke operation was used except that the drum and the ossicles were not removed. All the overhang from the attic and tympanic antrum was removed, as was any remnant of the annular ring. This left the upper portion of the tympanic membrane free and unattached, and at the completion of the operation this was folded over the ossicles. The operation was completed by fashioning the membranous meatus in a manner to meet individual requirements. The method in which the membranous meatus is used, instead of the flap as suggested by Bárány, is excellent in this type of case if the external canal is large. A vaselin pack is placed into the attic and antrum, and is allowed to remain in position for several days. After it is removed, the cavity may or

may not be packed, according to the apparent needs. This method has been used in five cases.

In the postoperative care in cases reported in this paper it was noticed that for the first week or ten days the condition appeared to be progressing very satisfactorily; then in all cases but one granulations began to appear in the cavity. These were disregarded, and the patient was sent away for from a week to ten days and asked to instill alcoholic drops into the ear. In the course of five or six weeks it has been seen that the granulations had contracted and that epidermization had nearly become complete. From this time on repair was rapid and complete. This happened in four of the five cases. In the fifth case, of a boy who was difficult to treat because of his fear and apprehension, the granulations were troublesome. Roentgen treatment was used in an effort to control the granulations and it was partially successful. This boy had been observed over a period of two years, and at the present time the cavity of the attic and tympanic antrum are perfectly epidermized, although there is some granulation just within the concha in the mastoid. The reason for this is, I believe, that in this particular case this was a cellular mastoid and not the smooth bone of a sclerotic mastoid. Because the granulations are organizing and the patient is somewhat older and more tractable, no great difficulty will be encountered in causing epidermization. But even the present result I consider satisfactory because the middle ear has been conserved and sealed off from the mastoid and the hearing is good. It has been my experience that, sometimes, too much in the way of treatment has been done to cavities after radical mastoid operations.

It will be noted that the symptoms ranged in duration from two and a half to forty years, and that all patients complained of a very foul discharge from the ear (table 1). One patient had definite attacks of vertigo, one had severe pain with acute exacerbations of the disease, and one had severe neuralgic pain with any damming back of the secretion. The physical findings were essentially the same in all cases. The operative findings were the same except in the last case, in which a more or less cellular mastoid was present that necessitated more removal of the mastoid process than the Stacke operation usually effects. Four of the patients had definite cholesteatomas in masses or in pearls in the attic and tympanic antrum. All had infected granulation tissue in these regions. All the patients have been seen at intervals for two years since operation, and the results remain good except for the boy who was mentioned before. He is observed for two or three days during vacations, and if improve-

ment is as rapid in the next interval as it has been in the last, the probabilities are that he will be completely cured.

TABLE I.

CASES IN WHICH THE MODIFIED MASTOID OPERATION WAS EMPLOYED.

Case	Age, Years	Symptoms, Duration	Physical Findings	Operative Findings
1	27	Oily flakes, foul discharge. Acute exacerbations. Little response to treatment. 24 years.	Attic defect, antrum intact, hearing good, small sclerotic mastoid.	Cholesteatomas in attic and antrum. Ossicles not involved. Sclerosis of mastoid.
2	48	Foul discharge, symptoms of fistula, attacks of vertigo. 26 years.	Attic defect, cholesteatomas, symptoms of fistula, small sclerotic mastoids. Could not tolerate local treatment.	Cholesteatomas. Erosion of horizontal canal. Sclerotic mastoid. Ossicles not involved.
3	21	Foul discharge, pain with acute exacerbations. 3 years.	Attic defect, too small for treatment. Foul discharge, drum intact, sclerotic mastoid, hearing good.	Infected granulations and cholesteatomas in pearls in antrum. Cholesteatomas in attic. Ossicles not involved. Sclerotic mastoid.
4	47	Neuralgic pain, foul discharge. Very apprehensive. 40 years.	Drum intact, small amount of foul discharge. Small defect into attic, small sclerotic mastoid, hearing good.	Granulations in attic and antrum. Ossicles intact. Sclerotic mastoid.
5	9	Foul discharge. 2½ years.	Drum intact, small defect into attic too small for treatment. Mastoid somewhat cellular, foul discharge, hearing good.	Partially sclerotic. Cholesteatomas in antrum. Infected granulations. Ossicles intact. Mastoid somewhat cellular around antrum.

Results: Cases 1 to 4—Excellent.
Case 5—Granulations near concha, attic and antrum epidermized. Patient seen at regular intervals.

CONCLUSIONS.

It is my opinion that the modified radical mastoid operation after the method of Bondy, offers a better chance for obtaining a good result than does the typical radical mastoid operation in certain selected cases. These patients have chronic discharge from the ear, with intact drums and good hearing. The ossicular chain and middle ear do not seem to be involved, and roentgenologic examination reveals a small sclerotic mastoid. The disease obviously is present in the attic and tympanic antrum and not in the middle ear.

BIBLIOGRAPHY.

Blackwell, H. B.: A Modified Radical Operation for Chronic Suppurative Otitis Media Based on Thirteen Cases. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 25:908-912, 1916.

Bondy, Gustav: Totalaufmeisselung Mit Erhaltung von Trommelfell und Gerhörknöchelchen. Monatschr. f. Ohrenh. 44:15-23, 1910.

Bryant, W. S.: The Radical Mastoid Operation Modified to Allow the Preservation of Normal Hearing. Tr. Am. Otol. Soc. 10:292-295, 1906.

Graham, H. B.: Taking Stock of the Radical Mastoid Operation. Tr. Am. Otol., Rhinol., Laryngol., 40:267-273 (March), 1931.

Heath, C. J.: The Cure of Chronic Suppuration of the Middle Ear Without Removal of the Drum or Ossicles or the Loss of Hearing With Ten Cases. Lancet. 2:353-357 (Aug. 11), 1906.

Körner, Otto: Die eitrigen Erkrankungen des Schläfenbeins. Nach klinischen Erfahrungen dargestellt. Wiesbaden, J. F. Bergmann, 1899, 153 pp.

Kopetzky, S. J.: The Modified Radical Mastoid Operation: A Critique. Tr. Am. Laryngol., Rhinol. and Otol. Soc., 15:458-464, 1909.

Lillie, H. I.: An Empirical Clinical Classification of Chronic Suppurative Otitis Media. Atlantic Med. Jour., 31:559-564 (May), 1928.

Richards, G. L.: The Socalled Conservative Mastoid Operation, With a Description of the Technic of Heath, Bondy and Siebenmann. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 20:578-594, 1911.

XXXV.

A REVIEW OF THE INTERRELATIONSHIP OF PARANASAL SINUS DISEASE AND CERTAIN CHEST CONDITIONS, WITH ESPECIAL CONSIDERATON OF BRONCHIECTASIS AND ASTHMA.*

JOHN G. McLAURIN, M. D.,

DALLAS.

It is now realized by most internists and otolaryngologists that there is a close interrelationship between various types of sinus infection and certain diseases of the lower respiratory tract, but it is highly necessary that the general practitioner should have a clear understanding of the importance of a prompt and thorough investigation of the sinuses in an effort to explain the cause of most of the chronic nontuberculous chest diseases. Much valuable time could be saved to the patient if a competent otolaryngologist was consulted early in such cases. The chest conditions that can be traced most directly to sinus disease are peribronchitis, tracheobronchitis, bronchitis, bronchiectasis, and many cases of bronchial asthma. Wood stated: "Evidence of secondary infection of lower portions of the respiratory tract from nasal sinusitis is too absolute to be denied. Such occurrences are too frequent to be overlooked." Hajek advised the eradication of sinus disease in preference to pilgrimages to resorts in an effort to relieve the catarrhal changes in the larynx, trachea, bronchi and lungs. Dean is to be quoted as follows: "In chronic bronchitis and bronchiectasis the first step in therapy is the examination of the upper respiratory tract for evidence of chronic infection." Smith, after reporting the study of a large group of cases from a thoracic clinic, stated as follows: "No condition of the chest should be finally diagnosed without a thorough search for the foci of infection in the head."

These statements and similar opinions have been voiced by many excellent observers who have carefully examined large groups of such cases in the best American and European clinics. It is not necessary here to give the detailed reports of the studies of these

*Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, 1935.

and those men that are sufficiently interested may easily satisfy themselves of the relationship between sinus disease and the chest affections already mentioned. I believe that even though the lung disease comes as a sequel to scarlet fever, measles, whooping cough or influenza, it is likely that a sinus disease exists in those cases where the trouble in the chest persists and is resistant to relief measures directed to the chest itself.

Without going into great detail I wish to enumerate the most probable routes by which infection reaches the chest from the diseased sinuses, as follows: (1) direct aspiration of infectious material into the trachea and bronchial tree—the so-called aspiration or droplet infection, (2) lympho-hematogenous extension, (3) hematogenous extension, and (4) direct continuity of tissue. In 1930 and 1931 I injected the sinuses of a number of patients with a 40 per cent solution of lipiodol in olive oil, according to the Proetz displacement method, and in from twenty-four to forty-eight hours was able to demonstrate by roentgenograms films of lipiodol in various parts of the bronchial tree. These experiments were carried out while the patients were going about their business, which showed that the larynx, spoken of by Jackson as "the watch-dog of the lungs," is not always effective in its effort to keep foreign materials from passing by it. The importance of the droplet infection route can be realized when it is remembered that in many patients lipiodol can be instilled into the trachea by dropping it into the pyriform sinuses from whence it will flow into the larynx, even though no local anesthetic has been used. If a foreign substance, such as lipiodol, can so easily slip by the larynx, then it is reasonable to assume that mucopurulent material, a product of the patient's own tissues, can more readily drop from the postnasal space and find its way into the bronchial tree. Spence has recently reported that by x-ray pictures he has also found lipiodol in the chest after he had injected it into the sinuses. Mullin, in reporting experiments on rabbits, states that groups of cases, for medical literature is replete with such reports after he had injected india ink into the nostrils he had found on autopsy that there was ink in the trachea and bronchi. Quinn and Meyer instilled large quantities of iodized oil into the nostrils of patients sleeping under morphin and sodium barbitol, and by roentgenograms taken the following morning found small quantities of the oil in both lower lobe bronchi.

The lymphohematogenous route of infection was definitely demonstrated by some excellent work done by Mullin and Ryder, and reported in 1920 and 1921. These men injected an emulsion of

india ink and tubercle bacilli into the sinuses of rabbits and cats, and later found the ink in the submaxillary and internal jugular lymph nodes, the larger lymph trunks, the great veins, the right side of the heart, and thence in the pulmonary arteries and peribronchial lymph nodes.

Infection from the sinuses to the chest by the hematogenous route probably is very frequent, but to my knowledge there has been no experimental work reported that would indicate how often the infection is carried directly through the blood stream to the lungs. Infection can likely reach the lungs from the nose by direct continuity of tissue, but I believe that the other routes of infection are much more important and are probably responsible for most of the secondary infections in the lungs. I believe Mullin was almost correct when he stated, "In cases where there is little or no escape of pus from the antrum into the nose, I think the lymphatic route is probably the path of infection par excellence. On the other hand, in cases where the nose is constantly full of pus, it is possible that inhalation may be more important than absorption." I would alter the latter part of his statement to read that in cases where the nose is constantly full of pus, it is very probable that inhalation is more important than absorption.

Statistics from various clinics would indicate that nasal sinus disease is present in from 55 to 100 per cent of all bilateral bronchiectasis. Dunlan and Skavlen stated that 73 per cent of their cases of bronchiectasis had sinus disease, and Clerf reported that about 82 per cent of his cases showed sinus pathology. Unilateral bronchiectasis is not associated so frequently with sinusitis. I believe that the association of bilateral bronchiectasis and paranasal sinus disease is almost constant, the apparent exceptions being due to an inability of the examiner to determine always the existence of sinus disease. Of course, bronchiectasis can be caused by foreign bodies, lung abscess and fibroid phthisis, but such cases are not being considered in this paper. In many instances the chest disease is much more apparent than the pathology of the sinuses. Patients having the milder forms of bronchiectasis have a chronically recurrent productive cough, but their general state of nutrition is likely to be excellent. The sputum need not be raised in large quantities nor the physical findings very noteworthy. The cough is usually initiated by changes in body position. Physical examination may show only a few moist rales. The typical advanced bronchiectatic patient periodically raises large quantities of purulent foul sputum, may have recurrent hemoptysis, and febrile attacks occur at intervals. Clubbing of the fingers is

usually present. The nutrition of the patient may still be good. Hemoptysis probably occurs more frequently in bronchiectasis than in any other disease of the lungs. However, the diagnosis is best determined by roentgenograms taken after lipiodol has been instilled into the bronchial tree. The x-ray pictures may show only slight dilation of a few of the bronchi with some peribronchial thickening in the milder types, but will show marked dilation and sacculation, and even cavity formation in the more advanced cases.

The constant discharge of infected material into the lower respiratory tract causes a chronic infection in the mucosa of the bronchi with a weakening of the wall. The cilia of the mucosa is gradually lost and stasis is favored, which in turn causes further progress of the infection. Extra bronchial pressure from enlarged peribronchial lymph nodes is also responsible for retention of secretion. Assuming now that the bronchi have been damaged by infection and secretion retained in them, there must be another factor to cause the dilatation. Hedblom showed that with every deep inspiration a difference of 10 to 20 cm. of water exists between the negative intrapleural pressure and the positive atmospheric pressure in the bronchi. Such a force is insufficient to cause a dilatation of a normal bronchus, but may be sufficient to cause dilatation of one previously damaged by infection. Coughing does not produce enough increase in the intrabronchial pressure to cause dilatation, for any increased pressure that develops with the cough is counterbalanced by a sufficient external pressure exerted throughout the chest by the chest wall itself and the abdominal muscles. Cough is important, however, because it spreads infectious material from one bronchus to another.

A careful history of these bronchiectatic cases will usually indicate that the trouble started rather early in life and that as a child the patient had very frequent colds, indicating flare-ups of chronic sinus disease, associated with a great tendency to cough. It may be true that the sinusitis followed measles, whooping cough, scarlet fever or influenza. Every case of bronchiectasis must have a very complete examination of the sinuses, including carefully taken stereoscopic x-ray pictures. Roentgenograms made after the injection of iodized oil by the Proetz method are frequently of great value, and pictures taken after the direct injection of some particular sinus may give much worth-while information.

The best treatment for bronchiectasis is preventive, which simply means that sinus disease must be recognized early in life and unrelenting efforts made to control it. There must be excellent cooperation between the parents and the physician. The parent must

be made to understand what are the probable sequelæ of chronic sinus disease, and the otolaryngologist must make a systematic and constant effort to clean up the sinuses in these cases. If bronchiectasis is well developed when the patient is first seen it is also highly necessary to clean up any sinus disease that may exist. Unless this is done no improvement in the chest can be expected. If the sinus disease can be controlled the patient is likely to show marked improvement, even though it is not possible to relieve the dilatation that has occurred in the bronchi. These patients frequently gain weight, and the periodic febrile attacks cease; the sputum diminishes in quantity, and there is even some recession in the clubbing of the fingers. Where there has been marked dilatation or sacculation of the bronchi it may be advisable to aspirate through a bronchoscope any material that is being retained. These aspirations may be done twice or once a week at first, and later once every two or three weeks until much improvement has been seen. However, aspiration will accomplish very little unless the patients use postural drainage several times daily, and this is sometimes difficult to make them do. Repeated introduction of iodized oil into the tracheobronchial tree has been highly advocated. Its beneficial effect in bronchiectasis may be explained in two ways: first, its presence tends to displace the retained secretions and promotes their expulsion, and second, the iodized oil when injected exerts a bactericidal action by slowly liberating free iodin. *In vitro* the oil has no bactericidal action, but *in vivo* it has been found that it causes a marked reduction of bacteria in the sputum.

Surgical procedures for relief of bronchiectasis are in the realm of the thoracic surgeon. Phrenicotomy and artificial pneumothorax are useless in collapsing large bronchiectatic abscesses, but thoracoplasty, according to Sauerbruch's method, and lobectomy are very valuable in some instances. These operations should be attempted only in the most extreme cases, for the mortality is from sixty to seventy per cent, in the hands of the best surgeons.

I would be remiss not to mention the importance of general hygienic measures and the proper adjustment of diet in all cases of bronchiectasis. In this regard much more can be done in handling the early cases in childhood. I think there is no doubt that the sinus disease is primary in nearly all of these cases, and dietary measures are of marked value in controlling sinus disease. It seems that many of these cases show a very low carbohydrate tolerance as evidenced by mucopurulent bridges from the septum to the turbinals and a rather constant postnasal discharge. All degrees of inflammatory change

may appear in the nose and sinuses, but most of these pathologic changes will be quite favorably influenced by prohibiting the intake of acid producing starches, sugars, candies, syrups, preserves, etc. Tonics in the form of cod liver oil, for its high vitamin and iodin content, and spintrate, because of its ability to increase carbohydrate tolerance in addition to its organic iodin content, are very valuable in treating the active sinus infection and preventing recurrences. As a matter of fact, I am inclined to believe that all of these cases are probably the result of some general constitutional derangement most likely dependent on faulty body metabolism. Biochemic errors are in turn very likely responsible for the faulty metabolism, and until we realize the great necessity of correcting the upset biochemistry that exists, by adjusting the diet and introducing the agents that are deficient in each patient, we will never succeed to any degree in handling these troubles.

The subject of asthma presents so many angles as to its mechanism, etiology and treatment that it is possible only to discuss in a short paper some particular phases of the subject and to limit such remarks to certain types of the disease. Asthma may be defined as a condition of dyspnea resulting from a neurosis causing spasm of the bronchial muscles associated with hyperemia and turgescence of the membranes of the small bronchial tubes and an exudate of mucus. At this time I shall limit my remarks to spasmodic bronchial asthma that may be classed as to their etiology as follows: (1) allergic, (2) infectious, (3) endocrine, (4) biochemical and (5) reflex.

It is likely that those cases of asthma apparently caused by infection, endocrine imbalances and biochemic irregularities should really be classified as allergic. I believe, too, that there is fundamentally a hereditary influence, that may be spoken of better as a congenital allergic tendency or predisposition, in all of the types mentioned, with the possible exception of the reflex type. We certainly cannot change the parentage of these allergic patients, but the question immediately arises as to what is meant in this instance by hereditary influence? If hereditary influence means that a patient is born with an instability of the autonomic nervous system easily influenced by biochemical errors or endocrine imbalances, then we have something more definite to work with, and we may hope to change some of these faults.

The allergic cases are usually classified as extrinsic or intrinsic, according to the nature of the cause. Asthma of the extrinsic type is caused by various pollens, animal emanations or danders, foods and many other substances. All intrinsic bronchial asthma is probably

infectious, some product of inflammatory reaction serving as the exciting allergin. Therefore, it can be readily seen that the so-called allergic types and infectious types belong to the same classification and should be termed as extrinsic and intrinsic allergic asthma.

There are three general plans that may be followed in handling these cases: (1) to avoid contact with or ingestion of the allergen to which the patient is sensitive, (2) to desensitize the patient to the offending allergen, and (3) to relieve the underlying cause of allergic sensitiveness, whether it be biochemical, endocrine, autonomic imbalance or what not. The ideal plan would be to find the cause of allergic sensitiveness and correct it, and I believe this can be accomplished rather frequently if very careful histories are taken and a most exhaustive examination is made. It is probable that much more time would be consumed but far better results would be finally realized. It must be remembered that an allergic patient may be sensitive to one allergen for a while and that desensitization to that allergen may be accomplished satisfactorily, but later that same patient will become sensitive to another or may be several more allergens. It is my belief that proper dietary adjustments to correct faulty biochemistry, well planned endocrine therapy to bring about a perfect hormone balance, and general efforts to stabilize metabolic processes will frequently correct this so-called allergic state in which we probably always have a predominance of parasympathetic control.

However, some special consideration must be given to these so-called "intrinsic" or "infectious" asthmas. These are the cases that usually show no skin reactions to extrinsic allergens. Chronic sinus disease can be demonstrated in practically every instance. Hyperplastic sinusitis with special involvement of the ethmoids is the frequent type of pathologic change. My observations are that hyperplastic sinusitis is the rule in young adult and more advanced life, but in children spasmodic asthma is frequently the result of purulent sinusitis, especially of the maxillary sinuses. Ferris Smith has been able to demonstrate sinus pathology in about eighty-two per cent of all asthmatic cases, and has arrived at the following conclusions: "(1) A very large percentage of asthmas depend on chronic sinus disease, (2) the asthma may result from a co-existing sinus disease and the presence of some extrinsic allergen, (3) many cases are not diagnosed because of inadequate methods of examination; lipiodol injection and suffusion are of great service, (4) surgery in asthmatic cases has been discredited because of the poor conception of the involved pathology and insufficient surgery for its removal, (5) in ethmoid

infections, complicated by bone involvement, it is not possible to totally remove the disease in all cases; infection may persist and polypi recur; all of the other sinuses may be totally cleared of infection; and (6) only the most radical surgery will be adequate." I also wish to quote Mullin as follows: "Given a case of bronchial asthma in which the sinuses are involved by hyperplastic processes and the nasal cavities contain polyps, one cannot expect that any prolonged amelioration of symptoms will follow any other treatment than the complete surgical removal of the hyperplastic tissues." Weille concluded after sinus surgery to relieve asthma that about fifty per cent of his patients had long continued favorable changes in their asthma. Some of his patients had very conservative operations, such as simple antrostomies, while other patients were submitted to the most radical sinus operative procedures.

The conclusions of many competent men could be given and very diverse opinions would be heard. It is my belief that there are some asthma patients who will be cured by rather simple sinus operations and some that will be cured by very radical and extensive sinus surgery, but there are others that will not be relieved by surgery to the sinuses regardless of how complete it may be. Why is this true? I think it depends upon whether or not the patient is sensitized in his bronchial mucosa to the products of the inflammatory process in his sinuses. We must determine in advance, if possible, if the patient is sensitive to his own inflammatory products, that may be called infectious allergens, whether they be bacteria, toxins or something else, if we are to prognosticate the influence of sinus surgery on the asthmatic state. I believe this can be done by one of several methods. A bacterin made by culture from secretion obtained from a sinus washing may be used in skin testing by scratch or intradermal plan. If the test is positive it is very suggestive that the patient is sensitive to his infection. If that plan does not indicate sensitivity of the patient, then a section of membrane from a diseased sinus (these cultures being grown best in brain broth or ascitic fluid) could be used for testing sensitivity. There is no reason why some of this bacterin could not be instilled into the bronchial tree to see what reaction would result, for in allergic reactions the sensitized tissues are supposed to contain specific allergic antibodies, and it is when the specific allergen comes in contact with tissues so sensitized that allergic reactions result. If a definite reaction in the form of asthma should result from intratracheal instillation of the bacterin it would strongly suggest a sensitivity to the sinus infection, but if there was no reaction it would probably mean nothing in regard

to sensitivity. Determining sensitivity will aid in arriving only at a prognosis as to the ultimate relief of the asthma, but the advisability of sinus surgery in these cases should not depend upon that entirely. Many asthmatic patients who have multiple sinus infection will be relieved of asthmatic symptoms for a long time after a successful operation on any one of the sinuses, but the asthma will finally return. I believe this is very suggestive of the patient's sensitivity to his sinus infection and should serve as an indication for further surgery to clear up all sinus infection if possible.

I would list the indications for sinus surgery in asthmatics as follows:

1. Sinus disease that would demand surgery, regardless of the existence of asthma.

2. Cases where the removal of polyps or simple methods of drainage and ventilation have helped the asthmatic state, but where such measures will not completely eradicate the sinus disease.

3. Cases in which recurrent attacks of rhinitis with exacerbations of the sinus disease seem to precipitate attacks of asthma.

4. Cases in which the patient has been proven sensitive to his own infection, but the local sinus pathology would not ordinarily require surgery.

5. Where sinus disease exists and it has been found that attacks of asthma can be relieved by cocainizing the sphenopalatine ganglion.

Reflex asthma is usually attributed to obstructive or pressure lesions in the nose or nasopharynx, such as adenoid hypertrophy, polyps, deviated septa, hypertrophied turbinates, and nasal synechiæ. Many asthmatics have been cured by relieving these defects. The explanation of reflex asthma as given by Sluder is probably correct. Irritation to the terminal sensory twigs of the sphenopalatine ganglion sets up impulses in that ganglion that are transmitted to the vagus, sympathetic and portions of the trigeminus. Bronchial spasm can be the result of this reflex. There is no reason why infection in the sinuses could not cause sufficient irritation in the sphenopalatine ganglion to bring about this same reflex with a resulting bronchospasm. This would serve as a good argument for doing thorough sinus surgery for the relief of spasmodic bronchial asthma of supposed reflex origin.

1322 Medical Arts Building.

BIBLIOGRAPHY.

Clerf, Louis H.: Bronchiectasis Associated with Disease of the Nasal Accessory Sinus: Etiology and Bronchoscopic Treatment of Bronchiectasis. Arch. Otolaryngol., July, 1927.

Clerf, Louis H.: Bronchoscopic Treatment of Bronchiectasis and Lung Abscess. Am. Rev. Tuberc., December, 1931.

Clerf, Louis H.: The Interrelationship of Sinus Disease and Bronchiectasis with Special Reference to Prognosis. Read at the Pan-American Medical Association meeting, Dallas, Texas, March 23, 1933.

Dean, L. W.: The Relationship Between Infections of the Upper Respiratory Tract and Pediatric Conditions. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1930.

Hajek-Heitger-Hansel: Nasal Accessory Sinuses. Vol. 1, page 88.

Jackson-Coates: Textbook on Ear, Nose and Throat. Pages 36-37.

Lilienthal, Dr. Howard (New York), Dr. Sauerbruch (Berlin), Dr. Baumgartner (Paris), Dr. Ascoli (Rome), Dr. Coquelet (Brussels), and Dr. M. Lagos (Barcelona): Treatment of Suppurative Bronchiectasis. Proceedings of International Congress of Surgery, ninth session at Madrid, Spain, April 16, 1932.

McLaurin, John G.: Chest Complications of Sinus Disease. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1932.

Mullin, William V.: A Review of Sinus-Chest Infections. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1932.

Mullin, William V.: The Interrelation of Allergy and Otolaryngology. Arch. Otolaryngol., March, 1932.

Mullin, William V.: The Relation of Paranasal Sinus Infection to Disease of Lower Respiratory Tract. Jour. Am. Med. Assn., September 4, 1926.

Mullin, William V., and Ryder, C. J.: Studies in Lymph Drainage of the Upper Air Passages and Accessory Sinuses. Laryngoscope, 31:138.

Mullin, William V.: The Nasal Accessory Sinuses as an Etiological Factor in the Production of Bronchiectasis. Tr. Am. Laryngol., Rhinol. and Otol. Soc., 1921.

Proetz, Arthur W.: The Displacement Method of Sinus Diagnosis and Treatment, St. Louis, 1931.

Quinn, Lester H., and Meyer, Ovid O.: The Relationship of Sinusitis and Bronchiectasis. Arch. Otolaryngol., August, 1929.

Smith, Ferris: Asthma: Its Etiology and Surgical Treatment. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, December, 1929.

Spence, Elbert L.: The Relation of Chest Infection to Sinus Disease. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1934.

Weille, Francis L.: Studies in Asthma: XVIII. The Surgical Treatment of Chronic Sinusitis in Asthma. Jour. Am. Med. Assn., January 28, 1933.

Wood, V. V.: The Paranasal Sinuses as Sources of Infection. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1928.

XXXVI.

CHRONIC LARYNGEAL STENOSIS.*

FRANCIS LEJEUNE, M. D., AND

NEAL OWENS, M. D.,

NEW ORLEANS.

Probably no condition in otolaryngology is associated with more danger and concern or gives a more accurate test of the ingenuity of the laryngologist than chronic laryngeal stenosis. This condition has always been perplexing, consistently resisting the best efforts to effect a satisfactory solution, and until a comparatively short time ago practically every case was an unfinished problem. Schmieglow states that not more than fifty years ago chronic stenosis of the larynx was considered incurable. As late as 1870 von Schroetter's treatment of this condition by means of dilatation with tin bolts and hard India rubber was considered revolutionary. O'Dwyer, in 1885, strongly advocated intubation and later reported a series of five cases which he had successfully treated by this method. Lefferts, in 1890, demonstrated his instrument for intubation in cases of laryngeal stenosis in adults. Progress in the treatment of chronic laryngeal stenosis during the past fifty years has been most encouraging. It is gratifying to know that a condition considered incurable fifty years ago may be treated today by modern methods with the expectation of satisfactory results, good function and a fair approach toward normality.

We shall limit our discussion to the type of laryngeal stenosis associated with extensive scar formation. Lynch estimated that about 90 per cent of his cases, caused either by disease or trauma, were of this type, and he called attention to the distinct difference between the type of scar resulting from trauma and that resulting from disease. The vast majority of cases of chronic laryngeal stenosis associated with massive scar formation result from destruction by disease, irritation from an intubation tube, a tracheotomy wound placed high in the neck, or irritation from trauma with an associated perichondritis. Scar tissue resulting from trauma followed by infection is

*From the Department of Otolaryngology, Graduate School of Medicine; the Department of Surgery, School of Medicine, Tulane University; and the E. E. N. and T. Hospital, New Orleans, La.

Presented at the Tulane Alumni Clinics, December, 1934.

far more extensive than that seen following a disease process. In our experience the most extensive stenoses have resulted from perichondritis following trauma. In these cases there is extensive scar formation in the inner posterolateral surface of the cricoid and thyroid cartilages with fixation of the arytenoid cartilages, web formation, hyperplasia, osseous and cartilaginous formations in the larynx sufficiently large to practically obliterate the lumen. The cicatrix following a disease process is usually less extensive and is commonly located in the region of the cricoid.

Lynah, Barwell, Lynch and others have obtained excellent results in many cases of chronic laryngeal stenosis by means of bouginage and subsequent intubation. Iglauer devised an ingenious method of introducing a folded rubber tube into the larynx in such manner that it lay between the arytenoids and the upper margin of the tracheotomy opening, and demonstrated that continuous pressure of the folded tube against the laryngeal stricture was quite effective. Schmieglow has shown that many of the cases with high tracheotomies with thickening of the cricoid will respond satisfactorily if a low tracheotomy is performed. Johnson reports two cases which did not respond favorably to ordinary methods of dilatation but which showed considerable improvement after treatment with heated bougies. Tucker states that operation for chronic stenosis of the larynx is indicated when conservative methods, especially dilatation by intubation and bouginage, fail to effect a cure. He reports cases successfully treated by laryngotomy with removal of scar tissue and subsequent packing of the raw area with gauze. The packing was removed at the end of two weeks and replaced by Jackson's laryngostomy cannula carrying a rubber tube dilator over the upright cannula.

Arbuckle treated some of the more extensive and resistant cases by applying a Thiersch graft to the lumen of the newly dissected tracheal canal. His operative procedure consisted of making a midline incision through the larynx and exposing the lumen. Scar tissue which had formed at the level of the cricoid was removed by dissection. After a sufficiently large lumen was established a Thiersch graft was applied to the resulting raw area, and was mounted on a cigarette shaped rubber balloon, which was later inflated to insure approximation of the graft to the denuded area. Following the above procedure the laryngeal fissure was closed in layers and the balloon removed in eight days by direct laryngoscopy. His first two cases were operated upon by the above method, whereas in his other cases his technic varied only in that the graft was applied over a sea sponge which was fixed by a retention suture. The sponge was also removed

by direct laryngoscopy at the end of eight days. One case was reported where the sea sponge was coughed up before the graft had taken. Figi reports one case of laryngeal obstruction which was thought to be neoplastic until direct visualization through a midline laryngeal incision revealed the presence of a cicatricial diaphragm. The scar tissue mass was excised and the denuded area covered by Thiersch graft, covering the cricothyroid membrane. The graft was placed over a gauze sponge and held fixed by means of a stay suture. A tracheotomy tube was inserted at the end of the operation. At the end of nine days the lower end of the laryngofissure was reopened in order to remove the gauze, at which time it was found that the graft had taken.

Chronic laryngeal stenosis reduced to its final analysis is one of tissue destruction and its complications: infection, overproduction of scar, contracture and distortion. The correction of this condition is proper management of wound healing. It is axiomatic that the most satisfactory wound healing results from surgically sterile incised wounds with closely approximated borders and conversely, the poorest results in wound healing are obtained in chronically infected wounds with gaping borders, continually subjected to irritation and infection. In the former, healing by primary intention occurs with the production of a minimum amount of scar and distortion, whereas in the latter, healing occurs by secondary intention with the production of an abundance of scar, contracture and distortion. A common example, as a comparison of the two types of healing, is seen in a surgically sterile incised wound of the hand which has been properly closed by suture and a lacerated infected wound which is permitted to heal by secondary intention. In the former, healing normally occurs in from seven to nine days and a fine, linear, pliable scar results which shows little tendency toward contracture and distortion; in the latter, healing time is variable, depending on the extent of the laceration and virulence of the infection, and a thick, extensive scar usually results with excessive contracture and distortion. In such instances in the areas involved the skin is loose and the subcutaneous tissues soft and pliable, and as a result the defect in healing by secondary intention is minimal. If an infected wound occurs in an area where the skin is not loose and the subcutaneous tissues are somewhat fixed and less pliable, then healing will require a longer time and more scar tissue and contracture will result. Because of the chronic infection present and the rigidity of the larynx due to its cartilaginous framework, healing in chronic laryngeal stenosis is analogous to the latter type. The treatment of chronic

laryngeal stenosis is unusually difficult, as it is a condition which can not be successfully treated by conservative methods. The lesion is one of destruction of a portion of the lining membrane of the larynx from infection or injury and infection resulting in scar formation sufficiently extensive to virtually occlude a segment of the laryngeal lumen. The problem is not merely that of producing a closure of the laryngofissure but of reconstructing the stenotic portion of the larynx. This must be accomplished by a technic which will result in epithelialization of the ulcerated laryngeal trough, in order to facilitate the production of a permanently patent laryngeal lumen with unimpaired physiologic breathing, the latter being necessary for normal quality of voice. The problem of effecting epithelialization of the laryngeal trough is one which presents no little difficulty, for healing of this area by normal processes means healing by granulation and subsequent scar formation with no improvement over that which one is attempting to correct. We feel that four factors are responsible for the difficulty in effecting satisfactory healing in these cases: (1) chronic infection from contamination by secretions from the mouth; (2) chronic irritation caused by an indwelling tracheotomy tube; (3) the unsatisfactory healing and epithelialization of the lining of a cavity due to the inability to obliterate dead space; and (4) the ridigity of the segment produced by the cartilaginous tissue comprising the anatomic framework of the larynx. It therefore seemed logical that the only treatment which would offer permanent and satisfactory results would be that of complete eradication of scar by proper dissection and subsequent epithelialization of the newly formed lumen without scar formation and contracture. Based upon factors above mentioned, skin grafting was considered to be a requisite in effecting satisfactory epithelialization of the laryngeal lining because: (1) Healing could be effected in a short time, thereby reducing to a minimum the amount of scar tissue, contraction and stenosis; (2) healing could be effected in the presence of mixed infection which is usually present in these conditions; (3) the lumen of the cavity to be grafted can be lined by applying a satisfactory skin graft; and (4) transplanted to the area are normal epithelial elements which later undergo transformations and ultimately become not unlike normal mucous membrane. In support of the above contentions, both cases herein reported give a history of having been treated, seven and four years respectively, by numerous conservative methods with no satisfaction whatever. On the basis of the above hypothesis it was decided that the procedure offering the greatest opportunity for a permanent and satisfactory correction for chronic laryngeal stenosis was complete dissection of all scar, gran-

ulation, cartilage and osseous tissues which obstructed the laryngeal segment involved followed by an immediate application to the denuded area of a Thiersch graft over a "stent." Two cases so treated are herein reported.

REPORT OF CASES.

CASE 1.—A white male, aged 12, was admitted for relief of a chronic laryngeal stenosis associated with massive scar tissue formation. About seven years prior to the time of his admission in April, 1934, he was in an automobile accident and received a traumatic wound of the trachea. The edema of the trachea resulting from this wound was sufficient to require a tracheotomy with implantation of a tracheotomy tube. During the intervening seven years between twenty-five and thirty operative procedures were performed with no satisfactory results.

Physical examination showed a tracheotomy opening which contained a tracheotomy tube at its lower angle and which extended up along the midline of the neck almost to the level of the thyroid cartilage as a narrow, contracted, slit-like fissure. Externally one could see that there was almost a complete stenosis of the larynx above the tracheotomy. Under ether anesthesia suspension laryngoscopy permitted thorough examination with laryngeal visualization. This vizualization revealed fixation of the arytenoids and a considerable amount of scar tissue about the arytenoid joints. No vocal cords were visible. The region below the vocal cords was involved in a mass of scar which was so extensive that little could be determined at this time.

A midline incision was made from the upper part of the laryngeal fissure to a point about the level of the lower third of the thyroid cartilage. The thyroid cartilage was incised in order to obtain sufficient exposure for the eradication of the scar tissue. All scar occluding the trachea was removed by sharp dissection, along with a considerable amount of granulation tissue, bone, and cartilage. There was sufficient osseous tissue projecting from the floor of the trachea to necessitate its removal by rongeur forceps. Following the removal of the occluding mass a normal sized tracheal lumen was established, producing a large denuded surface along the lateral walls and in the floor of the newly formed cavity, from the level of the vocal cords downward to a point below the cricoid cartilage. A stent of the newly formed cavity was immediately made in order to obtain an accurate impression of the entire denuded surface. The stent was then covered with a Thiersch graft, skin surface adjacent to the stent, and reinserted into the laryngeal cavity. Two retention sutures were taken through the skin borders on either side of the laryngofissure in order to hold the stent fixed. Care was used to fashion the stent in such manner that the intratracheal tube could be inserted below its lower border. Following the insertion of the tracheal tube a dressing was applied to the wound. No attempt was made to close the wound at this stage.

After a period of nine days the retention sutures were removed and the stent was lifted from the laryngeal cavity. It was seen at this time that the graft had taken satisfactorily. The stent was cleansed with soap, water, and ether, and immediately reinserted into its old bed in the larynx where, by constantly holding the lateral walls expanded, it was worn as a safeguard against contracture. The condition of the graft apparently remained normal. There was, however, a tendency in the early postoperative stage for the size of the lumen to contract as was manifested by the increasing difficulty in inserting the stent into the cavity. This

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1. Photograph of patient when first seen, showing narrow, contracted, slit-like opening of the laryngofissure. Above the superior border of the laryngofissure was found a laryngeal obstruction composed of scar, bone, and cartilage.

Fig. 2. Photograph of patient following thyrotomy, excision of scar, and implantation of skin graft. Note the enlarged cavity which resulted from excision of the scar. As a result of the application of the skin graft the size of the cavity was maintained without any contracture whatever.

Fig. 3. Photograph of patient showing neck after the tracheal lumen had been re-established and complete closure of laryngofissure had been made.

attempt at contracture reached an equilibrium after about two months and following this the size of the cavity apparently remained unchanged.

Following an interval of about four months after the application of the graft we felt that sufficient tissue adjustment had taken place to enable us to close the wound without risk of further complications and consequently made preparation to complete the construction of the trachea. Although the graft appeared to be stable and without evidence of ulceration or contracture we felt that it would be judicious to attempt to effect some type of physiological closure, producing all the associated changes resulting from surgical closure and yet maintain an open laryngofissure accessible for direct inspection. From experimentation it was found that the borders of the laryngofissure could be approximated with ease by gentle pressure with the fingers and that the resulting approximation, in effect, gave the same results as a surgical closure. Adhesive strips were placed across the laryngofissure in such manner that the skin borders were held in approximation and it was found that a physiological closure would be maintained which excluded all air and permitted no drainage of mucus or other secretion from the trachea. The adhesive fixation functioned satisfactorily and dressings had to be changed only once in twenty-four hours. Following the application of the above dressing the patient was able to breathe in a normal manner, clear the throat of retained mucus, and showed a definite improvement in the quality of his voice.

After observing the effect of the physiological closure for a period of approximately two months without any deleterious results, it was felt that surgical closure could be produced with safety.

Under ether anesthesia and with the aid of suspension laryngoscopy the larynx and treachea were carefully examined and found to be patent throughout. The grafted area was visualized from above and was seen to have extended up to the level of the vocal cords. The arytenoids were more normal in appearance and showed definite movement, a manifestation not seen in previous examinations. A 10 mm. laryngoscope was passed into the trachea with ease. The entire survey appeared to be satisfactory, so a surgical closure was performed. The 10 mm. laryngoscope was left in place as a guide during closure. An elliptical incision was made around the laryngofissure in such a manner that the skin flaps, raised by dissecting the skin medial to the incision, would fall inward and be approximated with ease and without tension to form the roof of the newly formed laryngeal section. The flaps forming the roof of the larynx were approximated by interrupted suture of 0000 chromic catgut placed subcutaneously in order to avoid penetration of the laryngeal cavity. A second layer of interrupted chromic catgut sutures was taken in the muscle and then the outer skin borders were approximated by means of three interrupted mattress tension sutures of medium silk worm gut. The outer borders were closed by interrupted sutures of 0000 silk worm gut.

The patient made an uneventful recovery and the wound healed by primary union. All sutures were removed on the seventh day. Following the anatomical closure the patient experienced no difficulty whatever in breathing, was able to cough forcefully, and the quality of his voice was improved. There was, of course, a decided improvement in his general spirit.

Case 2.—An adult, white female, aged 26, was referred for treatment of a chronic laryngeal stenosis of about four years' duration. In November, 1930, as a result of an automobile accident a lacerated wound was received over the chin. In obtaining patient's history, she emphatically stated there was no penetrating wound of the larynx or trachea. There was no pain, ecchymosis, or soreness of

the throat, dysphonia or dysphagia. Two weeks following the accident she developed a definite hoarseness and shortly thereafter a pronounced swelling appeared over the region of the thyroid cartilage. Dyspnea followed within a few hours and became so severe that a tracheotomy was performed and a tracheotomy tube was worn from this date. Perichondritis with abscess formation in the tissues of the neck lateral to thyroid cartilage on the left developed. The abscess was drained. From December, 1930, until June, 1931, she developed numerous deep-seated abscesses in the neck which were incised and drained. In February, 1931, she contracted a pneumonia complicated by a lung abscess and later by a severe attack of pyelitis. From June, 1931, to May, 1934, the patient was under constant treatment. During this time one operation was attempted for the eradication of scar tissue, which was unsuccessful. Treatment during this period consisted largely of topical applications.

The above patient was seen by us for the first time in May, 1934, and at that time examined under suspension laryngoscopy. The trachea above the tracheotomy opening was found to be almost completely closed. A massive amount of scar was visible on the posterolateral wall of the thyroid cartilage below the level of the vocal cords and involved the entire area of the cricoid cartilage and the first ring of the trachea. There was fixation of the arytenoid cartilages. No cords could be visualized. It was decided that the open stent method of closure was indicated in this case in order to obtain permanent and satisfactory results. The same technic was performed in the second case as that described in the first, the operation and after care differing in no respect.

During the reaction stage of anesthesia, following the final operative procedure an intratracheal tube had to be inserted at the lower angle of the wound due to a partial obstruction by mucus. The tube was removed the following day and no difficulty whatever was experienced subsequently. The patient made an uneventful recovery, following this interruption, until the eighth day. The sutures were removed on the seventh day and the wound had entirely healed except for a small fistulous opening at the lower angle of the wound, resulting from the insertion of the tracheotomy tube on the day of operation. On the night of the eighth day the patient died suddenly from pulmonary embolus. Postmortem examination through the tracheal fistula showed no evidence of any obstruction.

COMMENT.

The rationality of the technic advocated for the satisfactory restoration of a laryngeal segment in cases of chronic laryngeal stenosis is based on the proper management of wound healing. Following a principle of plastic surgery that scar tissue production and distortion of a given healed wound is in indirect proportion to the denuded area covered by skin graft or transplant and the rapidity of healing which results, it was thought that some form of skin graft was essential to obtain epithelialization of the laryngeal cavity without subsequent contracture of the lumen. A pedicle skin transplant was not considered for two reasons: (1) The area to be transplanted would require a pedicle transplant too large in size, and its removal would give lateral distortion to the neck; and (2) there would be a definite possibility of the lumen of the trachea becoming subsequently nar-

rowed due to the formation of fat beneath the pedicle transplant. A Thiersch graft was considered the best, because it can be more quickly formed; the certainty of a "take" in relatively infective fields is greater than in other types of grafts; and it is known from experience that this graft remains stable when placed in the mouth, a location which would subject a graft to more irritation than the trachea. It has been shown, too, that a Thiersch graft in a mucous membrane lined cavity gradually undergoes a change until it is not unlike mucous membrane.

Following the principle advocated by Gillies in the reconstruction of the lining of the newly constructed nose, a stent was made of that portion of the laryngeal cavity which was to be grafted. We felt that a stent was preferable to the sea sponge or gauze, as advocated by Arbuckle or Figi, as a much more accurate impression of the area to be grafted can be taken with the stent and as a result more intimate approximation of the graft to the denuded area may be obtained. A Thiersch graft was next removed, applied over the stent in the usual manner, and then inserted into its bed within the denuded larynx. The stent was held fixed by approximating the lateral borders of the laryngofissure by means of two retention sutures. At the end of nine days the retention sutures were removed and the stent was lifted out of the larynx with no difficulty, permitting direct inspection of the entire area grafted.

Following the immediate postoperative stage, the stent was worn for a period of four months in both cases in order to permit readjustment of the involved tissues before completion of the closure of the laryngofissure. This procedure was associated with no discomfort, and dressings were changed with ease and satisfaction. The action of the stent during this time served to make the graft more pliable, since it was in constant approximation with the graft and acted, as the result of the normal movement of the neck and of deglutition, to massage its surface. Our findings during this stage were not wholly in accord with Figi, who stated that there was little tendency toward contracture of the laryngeal cavity. We noted a definite tendency toward contraction, which was manifested by increased difficulty in reinserting the stent during the early stages. This, however, disappeared after the stent had been worn for about five weeks, due to constant stretching and massage of the tissues by its presence in the laryngeal cavity.

By the "open stent method" of grafting these cavities a therapeutic closure of the laryngofissure can be effected by approximating the lateral borders of the cavity and holding them in fixation by

means of adhesive plaster strips. This procedure gives a functional closure simulating closely a normal one and enables the patient to breathe normally, cough with sufficient force to clear the throat of mucus, and to show a definite improvement in the quality of the voice. This test is unquestionably of importance, for it gives one the opportunity to visualize the reaction of the graft to its new location and to the secretions which constantly bathe its surface. If the graft reacts favorably during a test period of six to eight weeks, showing no tendency to macerate or contract, we feel that one is justified in effecting a surgical closure, by layers, with little or no fear of subsequent ulceration or contracture.

The long time which is necessary to carry out the above technic is, in many cases, not a disadvantage, for a large number of these cases are poor surgical risks when first seen and require supportive treatment to rebuild their resistance. It is not improbable, after closely observing the results following the different stages in this technic, that the time required may, with safety, be greatly reduced.

632 Maison Blanche Bldg.
1430 Tulane Avenue.

BIBLIOGRAPHY.

Arbuckle, M. F. C.: Cicatricial Laryngotracheal Stenosis Treated Successfully by Open Operation and Skin Graft. Trans. Amer. Laryngol., Rhinol. and Otol. Soc., 33:450, 1927.

Barwell, Harold: Cicatricial Stenosis of Larynx Treated by Intubation. Lancet, 1:462, 1916.

Figi, F. A.: Laryngofissure and Skin Graft. Surg. Clin. N. Amer., 11:837, 1931.

Gillies, Sir Harold D.: Plastic Surgery of the Face, London, H. Frowde; Hodder and Stoughton, 1920.

Iglauer, Samuel: Some Original Methods of Treating Laryngeal Stenosis. Annals of Otology, Rhinology and Laryngology, 27:1233, 1918.

Johnson, W. H.: Use of Electrically Heated Bougie in Postdiphtheritic Cicatricial Stenosis. Annals of Otology, Rhinology and Laryngology, 38:1145, 1929.

Lefferts, George: Intubation of the Larynx in Acute and Chronic Syphilitic Stenosis as a Substitute for Tracheotomy and the Method of Schrötter. New York Med. Rec., 38:369, 1890. Quoted by Schmieglow.

Lynah, H. L.: Laryngeal Bouginage. New York Med. J., 110:838, 1919.

Lynch, R. C.: Stenosis of the Larynx. Trans. Amer. Acad. Ophthalmol. and Otolaryngol., 33:355, 1928.

O'Dwyer, J.: Congenital Stenosis of the Larynx. Trans. Am. Pediat. Soc., 9:180, 1897.

Schmieglow, E.: Laryngeal Stenosis: A New Method of Treatment. Arch. Otolaryngol., 9:473, 1929.

Tucker, Gabriel: Laryngostomy for Laryngeal Stenosis. Arch. Otolaryngol., 1:157, 1925.

von Schrötter, Ludwig: Quoted by Schmieglow.

XXXVII.

SUBCUTANEOUS EMPHYSEMA AS A COMPLICATION OF FOREIGN BODY IN A BRONCHUS.*

LOUIS H. CLERF, M. D.,

PHILADELPHIA.

The spontaneous occurrence of subcutaneous emphysema in a case of foreign body in a bronchus prior to bronchoscopy or other surgical procedure is an alarming complication. Subcutaneous emphysema of the neck, extending at times to the face and trunk, is not uncommon after tracheotomy. It is observed in wounds, injuries or surgical operations of the esophagus, the upper air passages, larynx, tracheobronchial tree or lungs. The development of subcutaneous emphysema as a complication of pertussis, diphtheria and pneumonia in children has been reported; it also was observed in cases of influneza during the epidemic of 1918.

In 1922, Wiskovsky and Camrda[1] reported the occurrence of emphysema of the mediastinal and subcutaneous tissues following aspiration of a bean into the right bronchus. Forbes[2] recorded the development of extensive subcutaneous emphysema in a child, three days after a peanut had been aspirated. In Woodward's[3] case, a child aged six years, subcutaneous emphysema occurred following the aspiration of a grain of corn. In one of three cases reported by Vinson and Moersch,[4] the foreign body, a paper clip, was believed to have penetrated the bronchial wall. In the remaining two the foreign bodies were peanuts. In both, there was emphysema of the subcutaneous tissues of the neck, head and upper thorax; in one, there also was air in the pericardial sac. In the case of a child, aged three years, reported by Carpenter,[5] emphysema occurred three days after choking on a peanut. The involvement was very extensive, including the scalp, face, neck and entire trunk down to the pubes.

In all of these cases the emphysema occurred prior to attempts at removal. In none was there an associated pneumothorax. In all the emphysema cleared promptly following bronchoscopic removal of

---

*From the Bronchoscopic Clinic, Jefferson Hospital, Philadelphia.

Presented before the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Norfolk, Va., January 19, 1935.

the foreign body and recovery was complete without mediastinal or pulmonary complications.

A fatal case of foreign body in a bronchus accompanied by spontaneous emphysema and complicated by a suppurative mediastinitis and pericarditis was reported by Imperatori.[6] Two days after choking on a peanut the child developed swelling of the tissues of the neck. This extended to the subcutaneous tissues of the chest and seemed to increase after each paroxysm of coughing. The findings by physical examination and roentgen study showed changes in the lower lobe of the right lung with subcutaneous emphysema of the head, neck and chest; also involvement of the mediastinal tissue. At bronchoscopy a peanut was removed from the middle lobe bronchus and an additional small fragment from the lower lobe bronchus. Postoperative improvement was temporary; the patient died about ten days later from suppuration involving the mediastinum and pericardium. At autopsy nothing was found to explain the emphysema. It was suggested that there was a break in the party wall between the trachea and the esophagus.

Two cases of bronchial obstruction produced by foreign bodies complicated by subcutaneous and mediastinal emphysema were observed by me. These are of interest because of the extent of the emphysema, the rapidity of its occurrence, the diagnostic problem involved in one, and the prompt clearing up of the emphysema following removal of the foreign body.

REPORT OF CASES.

CASE 1.—A child, aged 21 months, developed swelling of the left cheek secondary to a tooth infection. Five days later there was cough with fever (104°F). The family physician suspected measles. Fever persisted. On the twelfth day of the illness following a severe paroxysm of coughing there was noted swelling of the neck. This spread rapidly to the face and chest, being more marked on the right side. Dr. D. F. Daley was consulted and found evidences of partial obstruction to the left bronchus with subcutaneous emphysema of the face, neck and chest. On questioning the family he learned that the child had eaten a whole cooked prune at the onset of the illness and more recently had been given peanut candy. There was, however, no history of choking or gagging.

On admission to the Bronchoscopic Clinic, Jefferson Hospital, about twenty-four hours after the onset of the emphysema the child seemed comfortable when undisturbed. There was no dyspnea nor wheeze. The head, neck, shoulders and upper chest were enormously swollen. The eyes were completely closed (Fig. 1). The skin over the swollen areas appeared glossy, pallid and tense. Attempts to palpate or percuss the chest over the emphysematous area provoked crying, suggesting that this was painful. Dr. T. McCrae saw the child in consultation but could not make a satisfactory examination of the chest on account of the emphysema,

Fig. 1. Photograph of child aged 21 months, with extensive emphysema of tissues of head, neck, upper chest and mediastinum. The size of the head and neck was enormous in contrast to the remainder of the body. The eyelids were swollen completely shut. The quantity of air in the tissues was so great that characteristic crepitation could be elicited only along the junction of the normal and emphysematous tissues.

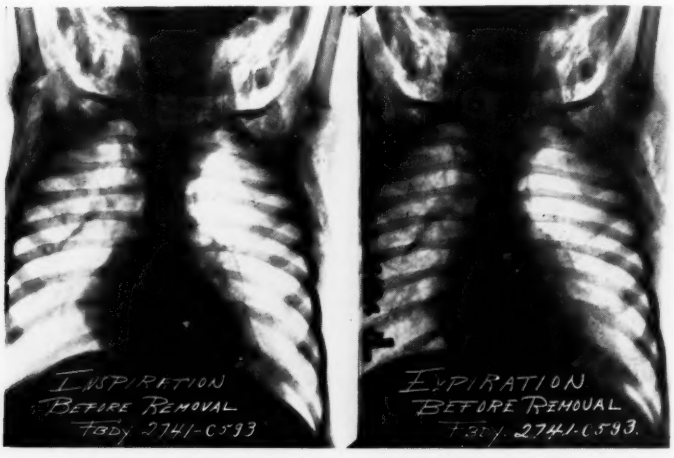


Fig. 2. Roentgenograms made at the end of inspiration and expiration show moderate obstructive emphysema of the left lung. The mediastinal emphysema is quite extensive. There is roentgen evidence of air in the tissues along the left border of the heart. The subcutaneous emphysema of the neck, about the shoulders and the lateral aspects of the chest is clearly shown. (Films by Dr. W. F. Manges.)

which extended to the fourth rib anteriorly, in both axilla laterally, over the upper back, and also because of the distress produced by movement of the child. The signs over the lower back, the only area that could be properly examined because of an absence of emphysema, indicated that there was partial obstruction to the lower lobe of the left lung. The roentgen findings reported by Dr. W. F. Manges corroborated this. In addition to the obstructive emphysema of the lower lobe there was observed emphysema involving the mediastinal tissues (Fig. 2).

It was noted that there was a progressive increase in the extent and degree of the subcutaneous emphysema following the admission of the patient to the clinic. Although bronchoscopy was believed to be hazardous, the findings by physical examination and roentgen study and the increasing subcutaneous emphysema were considered to be of sufficient importance to warrant its being done without delay. At bronchoscopy a portion of peanut was removed from the left main bronchus. It was observed at this time that there was marked forward bulging of the posterior pharyngeal wall, indicating a retropharyngeal accumulation of air; the trachea also was narrowed. The larynx appeared normal. Recovery was uneventful. The subcutaneous emphysema cleared promptly and the patient was discharged well ten days following bronchoscopy. A roentgen study at that time indicated that the mediastinal emphysema had cleared and that both lungs functioned normally.

CASE 2.—A child, aged 14 months, choked while eating peanuts. About twenty-four hours later, following a severe paroxysm of coughing attended with cyanosis there was noted swelling of the right side of the neck and face. On admission to the Bronchoscopic Clinic, Jefferson Hospital, two days following the accident, there was observed marked subcutaneous emphysema of the neck, head and upper chest, being more marked on the right side and over the upper end of the sternum (Fig. 3). The physical signs indicated that there was obstruction to the bronchus of the right lower lobe. This was corroborated by roentgen study; however, there was no obstructive emphysema present. There was roentgen evidence of emphysema of the mediastinal tissues. Following removal of the foreign body, a peanut, the emphysema promptly cleared.

COMMENT.

There was no roentgen evidence of pneumothorax in either case, although air could be demonstrated in the mediastinal tissues in both. The extent of the emphysema rendered physical examination difficult in case No. 1; the history was misleading, but the physical and roentgen findings were sufficiently positive to warrant a diagnosis of partial obstruction of the bronchus. The emphysema did not lessen in degree or extent until after the foreign body was removed. In case No. 1 there was an appreciable increase of emphysema up to the time of bronchoscopy.

The exact mechanism of production of emphysema in these cases is not known as none have come to autopsy. It is interesting to note that of the seven cases previously reported and the two cited herein eight of the foriegn bodies were of vegetal origin. These were incapable of producing direct trauma to the wall of the bronchus as

Fig. 3. Photograph of child, aged 14 months. Preminence of the air-containing tissues about the front of the neck and over the upper end of the sternum is clearly shown. The tissues of the right side of the face were more involved than those of the left. The right eye could not be opened.

might occur with a penetrating foreign body. In the case of paper clip in the bronchus with emphysema,[4] the pointed end was believed to have penetrated the bronchial wall. If this is the factor necessary for the production of subcutaneous emphysema it should be a common complication in cases of safety pins or other pointed metallic foreign bodies in the bronchi. On the contrary, it is rare.

An interesting study concerning the occurrence of subcutaneous emphysema in a group of fatal cases of pneumonia was reported by Clark and Synnott.[7] A total of twenty cases was observed. Of these, three were carefully studied at autopsy. The common distribution of the "gas" was about the base of the neck anteriorly and laterally and over the upper portion of the chest to about the third rib. In two cases it was more extensive, being distributed in the tissues of the cheeks and eyelids and in small areas over the sides of the chest and abdomen. At autopsy this distribution was corroborated. In the marked cases there was evidence of "gas" in the vascular sheaths of the arteries of the upper extremity extending as far as the thumb. It was also found in the mediastinal tissues, beneath the pleura at the hilum of the lung and in the smaller pulmonary septa. It was not observed in muscle sheaths. Small bits of fascia containing "gas" were found negative for aerobic and anaerobic bacteria. They reported that the air-containing alveoli appeared emphysematous in

the more nearly consolidated lung lobes. The distribution of the "gas" in the tissues corresponded with the dissemination one would expect as it escaped under pressure from the weakened alveoli of the lung.

The similarity in the distribution of the air in these and in the foreign body cases is striking. It is probable that the underlying mechanism is identical. Obstructive emphysema is a common clinical finding in vegetal foreign body cases. In the presence of a severe paroxysm of coughing distended air vesicles in a case of marked obstructive emphysema may rupture, forcing air into the lung tissues. The leakage of air lessens the degree of obstructive emphysema, but the valvular obstructive mechanism in the bronchus remains so that a certain quantity of air is forced into the tissues through the ruptured alveoli during each expiration and particularly when the patient cries or coughs. Air forced along the hilum of the lung into the mediastinum finds its way through the upper aperture of the thoracic cage to the tissues of the neck, face and trunk. The extent and degree of the subcutaneous emphysema are dependent in great part if not entirely on the effectiveness of the valvular action of the foreign body. With removal of the foreign body, air contained in the bronchi can no longer be forced through the ruptured alveoli and the emphysema subsides. The air in the tissues is promptly absorbed.

In conclusion, it can be stated that the fundamental requirements for the production of subcutaneous emphysema complicating bronchial foreign body are rupture of overdistended alveoli such as occur in obstructive emphysema through an increase in the intra-alveolar tension as is observed in paroxysms of coughing. Air entering a bronchus must be trapped during expiration. The degree and extent of the dissemination of the air in the tissues are dependent on the effectiveness of the valvular action of the foreign body. Prompt removal of the foreign body is indicated to prevent further extension of the emphysema and the possible development of complications. The occurrence of mediastinal suppuration results from forcing infected secretions contained in the obstructed bronchus through the ruptured alveoli.

1530 Locust Street.

BIBLIOGRAPHY.

1. Wiskovsky, B., and Camrda, L.: Mediastinal and Subcutaneous Emphysema After Inhalation of a Bean in Right Bronchus. Recovery After Extraction of Foreign Body by Peroral Bronchoscopy. Brastislau lekar. listy, 11:195, 1922.

2. Forbes, H. H.: Case of Emphysema of the Neck and Chest (Preoperative) Following Inhalation of a Piece of Nut. Laryngoscope, 36:192 (March), 1926.

3. Woodward, F. D.: Report on Year's Work in Endoscopy. Va. Med. Monthly, 53:299 (August), 1926.

4. Vinson, P. P., and Moersch, H. J.: Subcutaneous Emphysema Following Aspiration of Foreign Body; Three Cases. Minnesota Med., 14:654 (July), 1931.

5. Carpenter, E. W.: Colossal Emphysema of Upper Extremities. South. Med. J., 25:640 (June), 1932.

6. Imperatori, C. J.: Pericarditis as a Terminal Lesion in Mediastinitis: Report of a Case, Following the Inhalation of a Foreign Body in the Bronchus and Accompanied by Spontaneous Emphysema. Trans. Am. Bronchoscopic Soc., 1933.

7. Clark, E., and Synnott, M. J.: Influenza-Pneumonia Cases Showing Gas in Fascial Tissues. Am. J. Med. Sc., 157:219 (February), 1919.

XXXVIII.

A SYNDROME OF PAIN AND PARALYSIS ARISING FROM INFLAMMATIONS OF THE PREVERTEBRAL SPACE.*

ARTHUR W. PROETZ, M. D.,

ST. LOUIS.

There is a train of symptoms, encountered particularly during the inclement months, which center chiefly about a deep shoulder pain, usually impossible to locate by external palpation.

In a certain proportion of cases one sign occurs which has drawn attention to the prevertebral space as the source of the trouble, namely, a sore spot so located that a bolus of food passing down the esophagus aggravates the pain.

Before presenting the evidence which incriminates the prevertebral space it may be well to enumerate these related symptoms. They are:

1. Deep pain, as of a muscle spasm, an inch to one side of the spinous processes anywhere between the VII cervical and the III thoracic vertebræ.

2. Futile attempts to hold the head in some position which will relieve the deep cervical-thoracic tension; partial relief on lying supine.

3. Radiation of the pain to the arm, involving any or all of the branches of the brachial plexus.

4. Weakness and paralyses of various muscles attributable to brachial neuritis.

5. Occipital pain and tenderness.

6. A sore spot in such proximity to the esophagus that a bolus of food, passing down the tube, increases the pain.

7. Inflammation of the pharyngeal lymph tissues (or history of recent acute infection of these tissues).

8. Absence of fever or other severe systemic effects.

*From the Department of Otolaryngology, Oscar Johnson Institute, Washington University.

Presented before the meeting of the American Laryngological Association, Toronto, Ontario, May 29, 1935.

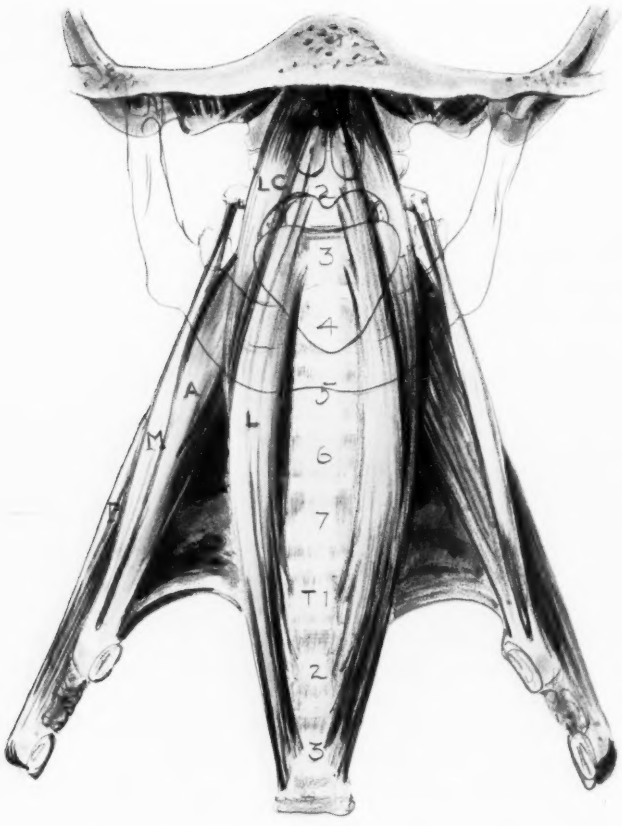


Fig. 1. Semi-diagrammatic representation of the prevertebral space, showing its relation to the spinal column, the Longus Colli muscles and the open mouth and pharynx. (Based upon an illustration from Spalteholz.)

L—Longus Colli muscle.
LC—Longus Capitis muscle.
A—Anterior Scalene muscle.
M—Media Scalene muscle.
P—Posterior Scalene muscle.

Between the posterior pharyngeal wall and the bodies of the cervical vertebræ are two layers of fascia. Behind the first lies the retropharyngeal space; between the second and the vertebral bodies is the prevertebral space.

The first of these potential rather than actual compartments has been the subject of numerous studies.[1,2,5,6,7] The second, with which we are concerned, has received little attention beyond the bare descriptions by the anatomists. Most of the standard texts fail even to differentiate it clearly from the retropharyngeal space, although a dissection of the region, especially a sectional study, makes the distinction apparent. Actually the prevertebral fascia, though quite thin, is constant and definite: a jet of compressed air projected against its edge in sagittal section so as to separate the tissues along their natural lines of cleavage delineates it readily enough. The drawings accompanying a recent article on the parapharyngeal space by Hall[5] show this region very clearly. The importance of the differentiation lies in the fact that the prevertebral space incloses the *MM. Longi Colli* and also extends laterally and posteriorly, following the vertebral bodies for some distance. (See figures.)

Gray[4] distinguishes the two spaces in the following description:

"Parallel to the carotid sheath and along its medial aspect the prevertebral fascia gives off a thin lamina, the buccopharyngeal fascia, which closely invests the constrictor muscles of the pharynx and is continued forward from the *Constrictor Pharyngis Superior* on to the *Buccinator*. It is attached to the prevertebral layer by loose connective tissue only, and thus an easily distended space, the retropharyngeal space, is formed between them. . . . The prevertebral fascia is prolonged downward and lateralward, behind the carotid vessels and in front of the *Scaleni*, and forms a sheath for the brachial nerves and subclavian vessels in the posterior triangle of the neck."

Lying between the prevertebral fascia and the vertebral bodies is the prevertebral space, occupied by a very loose connective tissue and the MM. *Longus Colli and Capitis*. It is limited above by the insertions of these muscles into the atlas and the *pars basilaris* of the occipital bone, respectively. Unilateral contraction of these muscles helps to turn the head toward the contracting side. Bilateral contraction bends the neck forward. When opposed by the erectors, these muscles participate in the action of pulling the head into the coat collar, as one will instinctively do in an attempt to keep warm.

Fig. 2. Diagram of a dissection of the deep muscles of the neck showing the distribution of Chicago Blue solution injected into the upper end of the prevertebral space. Inset, a photograph of the dissection.

L—Longus Colli muscle.
LC—Longus Capitis muscle.
A—Anterior Scalene muscle.
M—Median Scalene muscle.
P—Posterior Scalene muscle.
PS—Prevertebral space.
PF—Prevertebral fascia.
CS—Carotid sheath.

The lateral boundaries of the space are exceedingly indefinite. The prevertebral fascia extends laterally behind the carotid sheath to cover the *MM. Scaleni,* and the space behind it is gradually obliterated between these muscles and the vertebral bodies in close proximity to the cervical nerves, as they emerge from their foramina. Below, the space extends behind the upper esophagus into the posterior mediastinum.

Of clinical importance in the present instance is its contiguity above to the posterior pharyngeal wall and its lymphatic structures, the pharyngeal tonsil and the lateral bands, through which it may be exposed to infection. There is little doubt that the pharyngeal lymphatics provide an efficient barrier to infection and that bacterial penetration through the fascial layers is infrequent; but it does occur, as evidenced by the formation of retropharyngeal abscesses, and it is only a short step from the retropharyngeal to the prevertebral space.

Few reports are to be found[9,10] of experimental work in regard to bacterial invasiveness of various types of tissue, but there can be no doubt that non-pus-producing invasions do occur and that infections of the various fascial compartments do not necessarily eventuate in abscess formation.

An infection of the prevertebral space would have access to, and might involve:

The *M. Longus Colli*
the *M. Longus Capitis*
the pharynx
the esophagus
the posterior mediastinum
the vertebral bodies
the intervertebral ligaments
the spinal nerve trunks C I to T III

To verify this, a fresh soft cadaver was obtained and the head was sawn across, just below the level of the cribriform plate, so as to give access to the nasal chambers. With the body erect, a long needle was inserted from above into the prevertebral space as high as conditions permitted, and approximately one-half inch to one side of the median line. The needle was connected with a small reservoir containing a dilute solution of Chicago blue, which was elevated only ten inches in order to keep the fluid stream at a very low head. The fluid seeped into the space with comparative ease, six cc. being absorbed in slightly more than two hours.

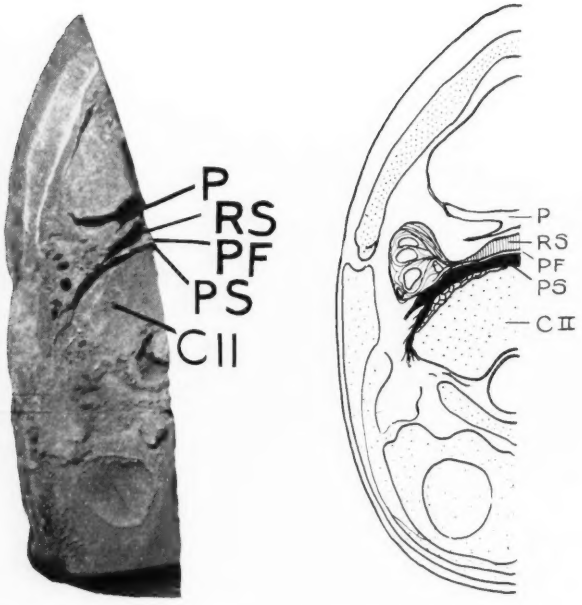


Fig. 3. Photograph and diagram of a cross-section of the neck at the level of the CII vertebra showing the relations of the retropharyngal space, the prevertebral fascia and the prevertebral space. Note the lateral extension of the space to the side of the vertebra in the neighborhood of the cervical nerves.

P—Pharynx.
RS—Retropharyngeal space.
PF—Prevertebral fascia.
PS—Prevertebral space.
CII—Second cervical vertebra.

The head and trunk were then cut in two, sagitally, and the injected half was sectioned transversely for study. As the fluid was found not to have crossed the mid-line, the remaining half was similarly injected, and dissected without sectioning.

It was found that the stain remained well lateral to the mid-line, following the prevertebral fascia caudad and laterally, and staining the structures enumerated above with the exception of the posterior mediastinum and the spinal nerve trunks, which it approached but did not reach.

It was demonstrated also by an injection beneath the mucosa of the face of the sphenoid that no direct continuity exists between this and the prevertebral space, the thick muscle insertions of the *MM. Longus Colli* and *Capitis* forming a barrier at least one inch wide.

Le Gros Clark[2] dropped a 10 per cent solution of potassium ferrocyanide and iron ammonium citrate in the nasal cavities of rabbits, which he killed at intervals of one, two and three hours, washed and fixed in formalin. In one of his rabbits, killed one hour after treatment, the solution was found to have reached, among other things, "the deeper tissues in the orbit and the cellular tissue among the nuchal muscles, even extending on to the occipital aspect."

It would not be unreasonable to regard this avenue of infection as a possible source of the "nodular headaches" described by Sluder,[11] of which he says "Nodules would appear under the skin of the neck, sometimes under the scalp, usually at the height of the pain. Their appearance followed the distribution of the lymphatic glands as given by Bartels. . . . the nodules lasted twelve hours, to disappear only as the attack subsided, which was the usual course. In other cases I failed to get such a response [i. e., through cocainization of the nasal ganglion]. They subsided only as the patient in general became better. The nature of these nodules is not known."

The spinal nerves whose roots correspond to the lateral limits of the prevertebral space, namely, the CI to the TIII, are distributed over wide areas to structures too numerous to tabulate here. Some of them are the sites of pain for which it is commonly difficult to account. Such, for example, are the occipital regions supplied by the Second, arising at the level of the pharynx, and muscles of the neck supplied by the Third and Fourth, and the various groups of shoulder, arm and hand muscles supplied through the brachial plexus.

In a series of twenty-two cases, the complaints were similar and the central pain did not vary in its character and location. Only

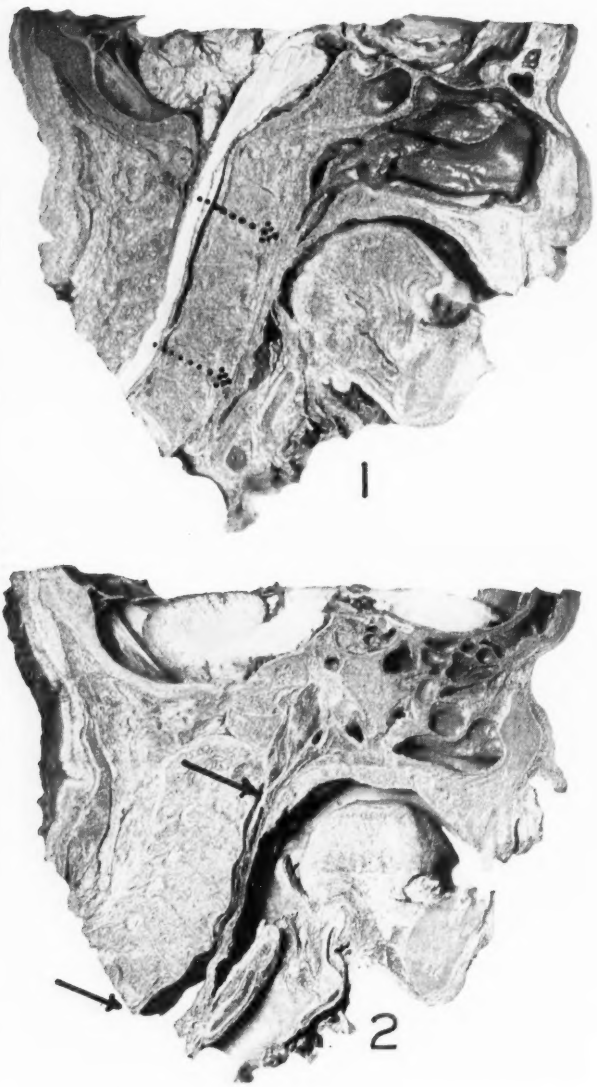
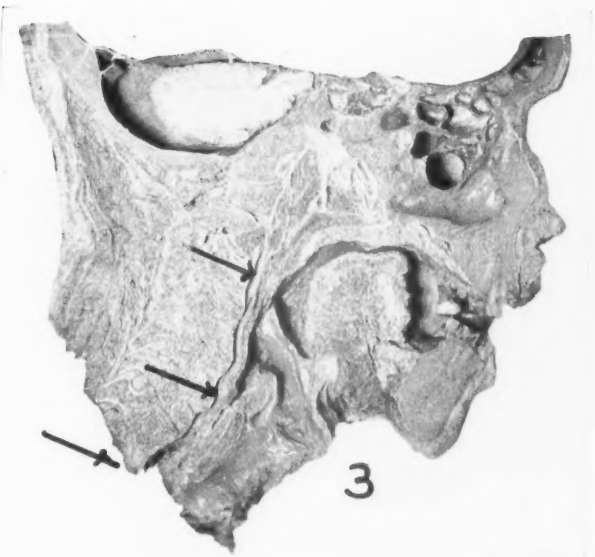
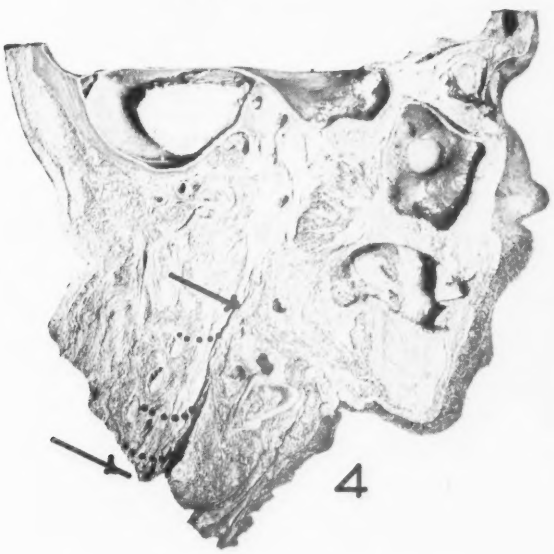


Fig. 4. Sections showing the relations of the prevertebral space. Section 1. Midline, showing obliteration of this space in the midline. Sections 2 and 3, more laterally showing relations of vertebrae and prevertebral space. Note the disappearance of the retropharyngeal space in 3. Section 4 shows the space ex-





tending lateral to the bodies of the vertebrae. The tissues in these sections were separated by means of a jet of compressed air, which reveals the natural lines of cleavage.

two of them presented the complete syndrome. The remaining twenty presented all but one or two sufficiently definite to include them in this group.

The most constant symptom is the deep pain in the region of the *M. Longus Colli,* characterized by the inability of the patient to localize it, or the examiner to elicit it on external palpation. Like most cervical myositis, changes in position afford little relief.

The exacerbation of pain when a bolus of food traverses the esophagus is sometimes not present, but it is extremely suggestive when it does occur.

Sensory disturbances of the brachial nerve branches are usually present. Motor paralyses are more rare. In three cases these were pronounced, practically incapacitating the hand for a period of three to six weeks. In one case definite burning sensations were described in the skin distribution of the posterior brachial cutaneous branch of the axillary nerve (origin V and VI cervical).

The reaction of inflammation of the pharyngeal tissues is apt to be slight and the local lymph nodes are not much swollen.

Thus far no cases of bilateral involvement have been encountered.

The condition is apt to recur with a repetition of symptoms characteristic of the individual case.

In only one case was it possible to observe an infection of this region at autopsy. In this case the infection, beginning in the left mastoid, perforated deep in the neck, crossed far down in the prevertebral space to the opposite side, dissected its way upward, finally penetrating the spinal canal with the vertebral artery beneath the oblique ligament of the atlas and eventuating in an extradural abscess at this point.

COMMENT.

The evidence for the foregoing is admittedly circumstantial. Since the condition seldom leads to fatal results, postmortem verification must be rare. In view of the close adherence of the symptoms to the anatomic background and to the findings in the cadaver after injection, it seems justifiable to associate them with the prevertebral space. While an inflammation of the retropharyngeal space might cause some of the symptoms, it could not involve the *MM. Longi Colli* nor could it reach the spinal nerve roots.

1010 BEAUMONT BUILDING.

REFERENCES.

1. Beck, August L.: A Study of Twenty-four Cases of Neck Infection. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 42:741 (Sept.), 1933.

2. Clark, W. E. le Gros: Anatomical Investigation Into the Routes by Which Infections May Pass from the Nasal Cavities into the Brain. Reports on Public Health and Medical Subjects, No. 54, London, 1929, p. 13.

3. Dean, L. W.: The Proper Procedure for External Drainage of Retropharyngeal Abscess Secondary to Caries of the Vertebræ. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 28:566, (June), 1919.

4. Gray, Henry: Anatomy of the Human Body (22d Edition). Lea & Febiger, Philadelphia, 1930.

5. Hall, Colby: The Parapharyngeal Space: An Anatomical and Clinical Study. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:793 (Sept.), 1934.

6. Mosher, H. P.: Deep Cervical Abscess and Thrombosis in the Internal Jugular Vein. Laryngoscope, 30:365 (June), 1920.

7. Mosher, H. P.: The Submaxillary Fossa Approach to Deep Pus in the Neck. Trans. Am. Acad. Ophth. and Otolaryng., 1929, p. 19.

8. Mudd, Stuart: The Penetration of Bacteria Through Capillary Spaces. I. The Motility and Size as Influencing Filterability Through Berkefeld Candles. J. Bacter., 8:459 (Sept.), 1923.

9. Mudd, Stuart, and Warren, Shields: The Penetration of Bacteria Through Capillary Spaces. II. Migration Through Sand. J. Bacter., 9:143 (March), 1924.

10. Mudd, Stuart and Mudd, Emily D. H.: The Penetration of Bacteria Through Capillary Spaces. III. Transport Through Berkefeld Filters by Electro-endosmotic Streaming. J. Bacter., 9:151 (March), 1924.

11. Sluder, Greenfield: Nasal Neurology, Headaches and Eye Disorders. C. V. Mosby Company, St. Louis, 1927, p. 289.

XXXIX.

MÉNIÈRE'S SYMPTOM COMPLEX: ENDOLYMPH DECOMPRESSION WITH SYMPTOMATIC IMPROVEMENT. REPORT OF A CASE.

SIDNEY N. PARKINSON, M. D.,

OAKLAND, CAL.

It seems of interest at this time to report a typical case of Ménière's syndrome and the symptomatic improvement produced by incising the saccus endolymphaticus on the involved side. This procedure is not new; in fact, it was reported nine years ago by Portmann[1] and since then has been performed by quite a number of otologists. It seems of sufficient importance and significance not to be overlooked in the current discussions of this disease. It may be significant when the nature of the disease is under consideration and it probably still has a place as a method of treatment.

In order to avoid the confusion of terms that exists in this field, I cannot resist suggesting Recurrent Aural Crisis as a descriptive name. The word crisis is preferable to vertigo because the process is definitely cyclic and involves both auditory and vestibular function.

There has long been much confusion as to what should and what should not be called Ménière's disease. Out of this confusion Dandy[2] has defined a clear-cut entity: "Ménière's disease . . . is a series of terrific attacks of dizziness, nausea, vomiting and tinnitus, grafted upon unilateral defective hearing. The deafness is progressive." One could not wish for a clearer definition. These attacks are so distressing that even the symptomless intervals are marred by the dread of their recurrence. The tendency seems to be for the process, whatever it may be, to continue for several years until the function of the involved ear is lost. Until the work of Furstenburg[3] relative to salt concentration in the tissues there was nothing known that would either bring on an attack or terminate one prematurely. The pathology is unknown. The etiology was equally unknown until Furstenburg's work indicated that the attacks may be due to retention of sodium chloride or to undue sensitivity of the labyrinth to salt.

This entity need not be confused with the more common conditions where patients have one or more attacks of vertigo with or

without nausea or vomiting, whether occurring spontaneously, or following trauma, or accompanying infection. In such cases there is at least a probable etiology, be it labyrinthine hemorrhage, irritation or infecton. Also, it is quite different from cases of intracranial tumor. Here the diagnosis is based on symptoms and signs too well known to warrant discussion.

Dandy and others leave no doubt as to the reliability of eighth nerve section in terminating the attacks. Of forty-two cases so operated upon, all were given complete relief from vertigo, nausea and vomiting, and partial relief from tinnitus. Similar surgery has been known and used since its development by Frazier[4] and Spiller more than twenty years ago.

A review of the cases of Dandy,[2] Coleman and Lyerly,[5] and Cairn and Brain[6] indicates that their cases have all been relatively late, with the hearing on the involved side so markedly diminished that its sacrifice at operation was a negligible consideration. Nevertheless, these surgeons have recently succeeded in obtaining equal relief by sectioning only the vestibular portion of the nerve, presumably all the vestibular fibers and as few as possible of the cochlear so as to preserve whatever remnant of hearing there might be. The follow-up on these latter cases will be interesting in regard to future hearing. As the disease apparently affects both hearing and equilibrium, sectioning only the vestibular portion of the nerve for symptomatic relief need not prevent continued deterioration of the auditory portion.

As a result of his experience with these cases, Dandy[2] stated some conclusions that are worth comment from the otolaryngologist's viewpoint:

1. "Ménière's disease has probably nothing to do with the internal ear. It is merely an explosion of function of the inner ear and is due to a lesion of the auditory nerve.

2. "A lesion of the inner ear could not affect both the vestibular and auditory divisions of the eighth nerve.

3. Although many kinds of treatment have been suggested, "nothing has been of the slightest benefit until the recent advent of this operation (sectioning the eighth nerve)."

The work of Portmann[1] and the more recent work of Furstenburg[3] strongly suggest that the disfunction is in, or at least includes, the internal ear rather than being solely a "lesion of the auditory nerve." Cunningham's Anatomy[7] states that "The cavities of the

utricle and saccule are indirectly connected, and thus all parts of the membranous labyrinth are in communication with each other, and endolymph is free to move from one portion to another." Obviously then a "lesion of the inner ear" could very well "affect both the vestibular and auditory divisions of the eighth nerve." As to the third conclusion, it may be said that aside from the work of Furstenburg, endolymph decompression, as recommended by Portmann in 1926, has benefited many patients afflicted with this disease, although it cannot be said to be as effective or as prompt in results as nerve section.

Portmann's theory is essentially that crises are due to cyclic changes, chemical or physical, in the endolymph which circulates throughout the internal ear. He compares the condition to the ocular phenomena in glaucoma. The ductus endolymphaticus arises from the saccule and utricle, and through its termination in the perforated saccus establishes drainage connection between the endolymph system and the meningeal lymphatics. This drainage mechanism might be the site of disturbance. If so, increased facility for endolymph circulation should modify or terminate the crises. At the seventy-eighth session of the A. M. A., Otologic Section, Portmann reported several successful results following saccus incision, as also did Eagleton in discussion. The operation was said to be not difficult and to cause no undesirable after effects.

REPORT OF A CASE.

E. H., a dentist, aged 36, was referred to me September 14, 1932. He had suffered from recurrent attacks of vertigo, nausea, vomiting, and tinnitus for more than a year. The tinnitus was referred to the left ear and there had been progressive loss of hearing on that side. There had been slight tinnitus for four or five years preceding this. The crises had been irregularly spaced at first and relatively infrequent, but had become progressively more frequent until for three months they had come almost regularly each third day. The attacks were severe and distressing and incapacitated the patient for the entire day, necessitating an average absence of two days a week from his practice, with serious economic loss. Quite naturally he was becoming both alarmed and despondent. He had consulted many physicians and had had thorough examinations in search for a cause. Among various types of treatment he had had many ear inflations, and soon after the onset had had a complete antrum operation on the right side. Nothing had given relief.

Examination: General condition as determined by an internist was essentially normal. The tonsils were absent. The nose and sinuses appeared negative. The ear drums were moderately retracted. Audiometer showed 20 per cent loss left ear, most marked for high tones; the right ear showed slight loss. No reference in Weber test. Romberg and pelvic girdle tests were negative. Cold caloric test showed nystagmus after two minutes each side but of shorter duration on left side

and without nausea. During attacks there was a vigorous rotary nystagmus to the left.

In consultation, Professor Towne of Stanford Medical School obtained negative neurologic findings except for eighth nerve as stated. He advised complete rest for a month before considering surgery. This advice was taken but the crises continued unchanged. As hearing was still useful he was reluctant to section the eighth nerve, although he felt that the ear would gradually lose acuity anyway if the process were not checked. He therefore advised Portmann's endolymph decompression operation as the next step, stating that he would perform nerve section if this failed.

Further study and corroboration were obtained from Albion Howell of Berkeley, who had reported a similar case at a local hospital staff conference.

During the four months that I observed the patient, typical violent crises continued to occur on an average of twice a week. This made seven months of frequent severe attacks preceded by a year of less severe and irregularly occurring ones, all in spite of constant medical attention.

Operation: January 5, 1933, increased drainage of endolymph was established on the left side by elevating the dura from the posterior surface of the petrous pyramid to the region of the fossa endolymphaticus and then carefully cutting across the saccus. Immediate postoperative recovery was uneventful and the patient returned to work at the end of one week.

Postoperative course (two years): Certain very favorable changes were apparent at once: (1) decreased tinnitus; (2) vertigo reduced to mild feeling of unsteadiness lasting an hour or so; (3) absence of nystagmus, nausea, and vomiting. Thereafter, for a year, there occurred what might be called "dizzy spells," in that at irregular intervals there would be slight increase in tinnitis and a sensation of unsteadiness not sufficient, however, to interrupt him at his work. As months went by the spells became steadily less frequent, encouraging us to believe they might finally cease. The last occurred at about one year from date of operation.

One year postoperative, audiometer showed further hearing loss left ear of 5 per cent. The reaction of the static labyrinth seemed unchanged. From the standpoint of actual use the hearing had seemed improved, due probably to decreased annoyance from tinnitus.

Two years postoperative, audiometer showed still further loss left ear of 20 per cent in spite of freedom from symptoms with the exception of mild tinnitus. Also, the left labyrinth showed definitely less reaction to the cold caloric test. These functions will be watched with interest in the years to come.

Clearly here is a case of Ménière's syndrome, the symptoms of which were distinctly and favorably affected by endolymph decompression. Doubtless many others could be reported. The very fact of effect from such a procedure suggests disturbance in the endolymph system. It seems reasonable that this may be of significance in study aimed at the etiology and pathology of the disease. It seems quite consistent with the work of Furstenburg, which indicates that the pathologic process is concerned with undue retention of sodium

chloride in the tissues. These local tissues are thought by him to have either an "increased avidity for salt or an unusual sensitivity to it. In not one instance," he states, "did we fail to produce an attack by the administration of sodium chloride and not once were we disappointed in obtaining complete relief by the medical therapy described."

SUMMARY AND CONCLUSIONS.

A case is reported of so-called Ménière's syndrome. The descriptive term Recurrent Aural Crisis is suggested.

Endolymph decompression, as described by Portmann, was performed on the involved side. The effect was to promptly and favorably modify the course of symptoms sufficient to rehabilitate the patient.

Continued deterioration in auditory and vestibular function indicates that the disease process has not been terminated in spite of symptomatic relief.

The relation of endolymph decompression to the symptomatic course of this disease may be significant in studies aimed at etiology and pathology. It seems consistent with Furstenburg's work on salt retention. It suggests that the endolymph is concerned in the pathology of the disease and in the production of the characteristic attacks.

This case is reported not with the idea of recommending the procedure used, but because it was used, and the effect has seemed interesting and instructive.

MEDICAL BUILDING.

REFERENCES.

1. Portmann, Georges: Vertigo; Surgical Treatment by Opening the Saccus Endolymphaticus. Arch. Otolaryng., 6:309, 1927.

2. Dandy, Walter E.: Ménière's Disease. Arch. Otolaryng., 20:1 (July), 1934. Menière's Disease; Diagnosis and Treatment. Report of 30 Cases. Amer. J. of Surg., 20:693, 1933.

3. Furstenburg, A. C.: Ménière's Symptom Complex Medical Treatment. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:35 (Dec.) 1934.

4. Frazier, Chas. H.: Intracranial Division of the Auditory Nerve for Persistant Aural Vertigo. Surg., Gyn. and Obst., 15:524, 1912.

5. Coleman, C. C., and Lyerly, J. G.: Ménière's Disease. Arch. Neurol. and Psychiat. 29:522, 1933.

6. Cairns, Hugh, and Brain, W. Russell: Aural Vertigo. The Lancet, 1:952, 1933.

7. Cunningham: Text Book of Anatomy, Wm. Wood & Co., Fifth Edition, p. 846, 1921.

XL.

THE PHYSIODYNAMICS OF INTRAVASCULAR FLUID INJECTIONS FOR ROENTGENOGRAPHIC STUDIES.*

O. Jason Dixon, M. D.,

Kansas City, Mo.

The chief clinical problem, which I wish to discuss, is a method of visualizing, with the x-ray, the intracranial venous sinuses by the injection of an inert opaque medium.

About four years ago, while carrying out some experimental studies upon vascular repair in the dog,[1] I resorted to the x-ray, in order to study more completely the vascular healing processes without disturbing the wound. As these surgical researches were concerned primarily with studies on sigmoid sinus thrombosis, the visualization of the injured vessel by x-ray promptly suggested the use of an opaque medium and the x-ray as an aid in the diagnosis of sigmoid sinus thrombosis.

Every otologist has at times been confronted with the perplexing problem, not only of determining whether the sigmoid sinus is thrombosed, but, when both ears are infected, which sinus is involved.[1a]

Trendelenburg[1b] was probably the first to suggest vasographic studies when, in 1902, he observed, by x-ray, a moving bullet within the heart chambers. Reithus,[2] Franck and Alwens,[3] and Schepplemen[4] made intravenous injections of various types of opaque medium in animals, which they studied under the x-ray. They failed, however, to develop a satisfactory medium for human use. In 1920 sodium iodid was used by Berberich and Hirsch,[5] Brooks[6] and Santous.[7] The latter was one of the first who attempted to visualize the vascular lesions in the brain by the injection of sodium iodid directly into the carotid artery.

---

*Read before the Dallas meeting of the Midwestern Section of the American Laryngological, Rhinological and Otological Society, January 28, 1935, at Dallas, Texas.

From the department of experimental medicine of the University of Kansas School of Medicine.

Sodium iodid produces a beautiful roentgenographic shadow, but when used in sufficient concentrations to produce this shadow it is so severe a vascular irritant that great harm results. I was immediately impressed with this danger when all of my experimental animals died within thirty-six hours after these injections. Even when only five cubic centimeters of a 50 per cent solution of sodium iodid was injected into the sublingual vein of the dog such a marked vascular irritation occurred in the lungs that the dog promptly developed an extensive pneumonia with pulmonary edema and pleural effusion. Obviously this medium is unsatisfactory for human use. Finally, in 1930, Radt[8] introduced a preparation called thorotrast,[9] which is a solution of thorium dioxid with an added colloidal substance which he used in the visualization of the liver and spleen. Allen and Camp[10] have recently reported successful arteriographic studies with this medium. As it is stored indefinitely within the reticulo-endothelial system of the various organs, its eventual harmful effects are undetermined. That it is not without some dangerous toxic reactions is shown by the fact that it has not yet been approved by the Council of Pharmacy and Chemistry of the American Medical Association.

About this time a nontoxic, inert iodin compound was sent to me for experimental studies. This substance had not yet been placed on the market, and was devised solely for intravenous injections for urographic studies. In certain amounts and concentration, it has been proven to be harmless and is quickly and completely excreted by the kidneys. For these vascular studies it fulfilled all of the necessary requirements except one: it did not show a complete shadow of the injected vessel. A faint line along the center of the channel was all that could be identified. This failure was due to the low viscosity of the solution, the center flow and rapid dilution within the blood stream, which will later be explained. Although it contained 52 per cent of inert iodin, it did not produce enough of the contrasting shadow. The animal was in no way disturbed by the intravenous injection of the solution, even in large doses, given at frequent intervals, but it became apparent that other factors played a part in the successful technic of the procedure. Certain factors were conceived and have been proven as necessary for satisfactory visualization and use in the human, and it is along the lines of physiodynamics and chemistry that certain phases of the problem are now being studied. The factors which play such an important part are as follows:

1. The opacity of the solution.

2. Complete filling of the channel that is to be studied with this opaque solution.

3. Limited miscibility with the blood in the region of investigation.

4. Immediate dissipation by gradual miscibility and complete elimination at some more distant point.

5. A viscosity, which will permit ease of injection, yet prevent rapid dilution by the blood.

6. A solution which will itself remain true to its purpose without chemical change, deterioration or otherwise become useless by the process of sterilization. The solution must be nonirritating to both the vascular endothelium and surrounding tissues, produce no harmful pathologic changes when mixed with blood, and should be harmless to all organs and free of all functional disturbances.

For injecting the sigmoid sinus, the sinus should be exposed in the usual manner following the mastoidectomy, and the solution injected directly into the blood stream, the needle being inserted distal to the site of the suspected lesion. The needle should be large enough to permit the chilled solution of glucose and opaque medium to enter rapidly but not so large as to make an unnecessary tear in the sigmoid sinus wall. The syringe should be equipped with finger grasps and the needle either screwed or locked in place in order to avoid obvious accidents. The bevel of the needle should not be too long, and in some instances the needle should be bent at an angle of 30 degrees, so that the edges of the mastoid wound do not interfere. The x-ray picture must be taken while the solution is flowing in and just prior to the last one or two cubic centimeters of injection. Care should be taken that none of this solution escapes into the mastoid wound because of the resulting confusing shadows. As the examined sigmoid is farther from the plate, any slight angulation may produce a confusing shadow (Fig. 1), so that interpretations should be made accordingly. Stereoscopic films make it much easier to differentiate the vascular shadows and should be used when possible. Care should also be taken to keep the hands of assistants and the operator out of the field, as finger shadows are easily confused with vascular shadows. Better pictures are secured with a patient under anesthesia while the mastoidectomy is being performed, but, with a co-operative patient, they may be taken without any anesthesia whatsoever, as there is no pain connected with the procedure.

I believe that the use of this contrast medium for the differential diagnosis as well as the definite diagnosis of the intravascular lesion within the sigmoid sinus, offers our first positive means of determining just what has taken place within this vessel and establishes a

Fig. 1. Postmortem injection of opaque medium directly into the opened sigmoid sinus. The patent internal jugular vein is clearly shown filled with opaque medium. It appears to be misplaced because of the malposition of the head.

definite method of procedure based upon more accurate preoperative evidence. Frenckner[11] has reported some experimental studies, which have recently been published. His work was carried on independently of mine, and, as he states, he has not used his method upon the living human. He also used thorotrast, which has the disadvantages previously mentioned.

I am now working with a chemist in the development of this opaque medium and I find that there are about thirty-five ways in which this chemically inert substance may be compounded. The development of a particular application of this medium has come about by combining it with glucose and then chilling. Various factors still remain to be worked out, and while I feel certain that the method that I have now used is safe, yet there remain some objectionable features which I hope to soon have removed and will report in a separate paper.

THE COMPOUND AND ITS PROPERTIES.

The solution which I have prepared and found to be the most satisfactory is a mixture of glucose and inert iodin compound of sufficient opacity and viscosity to fulfill the deficiencies of solutions previously used.

This solution contains approximately 19.2 grams of iodin per 100 cubic centimeters as combined iodin. This iodin is in chemical combination with carbon, hydrogen, oxygen and sulphur, in the form of a sodium salt. This iodin compound, with 71.5 grams of glucose in water solution, sufficient to make a total of 100 cubic centimeters, constitutes a solution of proper viscosity and opacity when used at a temperature sufficiently below body temperature to give intravascular viscous flow.

The purpose of viscous flow is to prevent rapid dilution of the opaque medium within the blood stream. Obviously the greater the concentration of the opaque medium near the obstructive lesion the more detailed will be the x-ray shadow.

The choice of a chilled and more viscous medium is unique in its application in that it contradicts the former practice of using a warm and less viscous solution. The former practice was carried on under the misapprehension that fluids injected into the blood stream at a temperature lower than that of the body were injurious. My former erroneous idea was that a less viscous solution was necessary in order to permit a rapid injection and thorough mixing of the opaque medium with the blood at the site of inspection.

Chilling of the solution was determined quite by accident. It was necessary to haul these animals a mile from our experimental laboratory to the x-ray room where these studies were made. In order to conserve time, my assistant filled all the syringes separately with the opaque solution and placed them in an open car, while we anesthetized the dogs with amytol and morphin, preparatory to the x-ray procedure. The solution, which was in the syringe, was in the open air for about one hour, and must have been only a few degrees above freezing, and at the time of the injection was probably between 40 and 50 degrees F. Although accurate temperature of the injected solutions were not made at this time, the Weather Bureau has since sent me a report made on that date, November 10, 1932. The maximum temperature was 36 degrees F. and the minimum temperature was 28 degrees F. When the intravenous injections were started in the usual manner this accidentally chilled solution had become so viscous that it was almost impossible to inject it into the sublingual vein. In fact it went in so slowly, and we were so certain of poor results, that we did not develop our films that evening. It was a very pleasant surprise, the next morning, to find that these were the first good films showing complete intravascular details with this inert and harmless type of solution. At once it was apparent that the lowering of the temperature with increasing viscosity was the important factor in the success of the procedure.

By referring to the viscosity temperature table for this solution, the optimum temperature for each desired use may be used.

SOLUTION VISCOSITY TEMPERATURE TABLE.

	No. 1.		No. 2.					
Temperature	20 per cent by Weight	40 per cent by Weight	Wt. %	25°	30°	35°	40°	50°
0° C	38.18	148.2	34.94	33.3	28.8	24.7	21.6	16.9
10° C	26.62	98.30	42.33	52.4	44.0	37.4	32.2	24.3
20° C	19.67	62.23	49.33	88.2	72.2	60.2	50.9	36.9
30° C	15.10	43.93						
40° C	11.97	32.61						

It is a well known scientific fact that any variation in temperature produces a corresponding variation in viscosity of any solution or liquid. The importance of viscosity was not fully appreciated, and although glucose was used in the attempt to fulfill this requirement, yet it was not until the glucose was chilled by accident that a proper viscosity was obtained, thus proving that the temperature of the solution plays a very important part as a factor in obtaining good x-ray results.

PHYSIODYNAMICS OF INTRAVENOUS INJECTIONS.

Although the intravascular injection of foreign substances is a very old procedure, there are still certain technical factors which are not thoroughly understood. It has finally remained for the physicist to develop certain accurate mathematical laws governing the flow of fluids. Obviously the physiodynamics of this problem, which we are now discussing, becomes a very important factor in the proper application and the correct interpretation of these intravascular studies, which are to be made by the use of this intravascular medium.

I have been fortunate to have the assistance of Dr. Robt. M. Isenberger, professor of pharmacology, University of Kansas School of Medicine, and Mr. E. R. Harrouff, Kansas City, whose training in chemical engineering and physiological chemistry has especially adapted him to the interpretation of some of these studies. Before entering into the highly technical phases of physiodynamics it may be well to describe just what is desired by the injection of this opaque medium into the blood stream. Granted that the previously mentioned objectionable factors have been removed, the complete accuracy of the studies of the suspected intravascular lesion is dependent upon the opaque medium completely filling the vessel throughout its course, so that any narrowing or deformity from its normal contour can be plainly seen in the x-ray shadow.

In a more detailed scientific study of the problem we are at once confronted with straight line and turbulent flow, and the velocity at the point of change of these two flows, which is called the critical velocity. The equation of Poiseville's law, for straight line flow, and Fanning's equation for turbulent flow, are used for mathematical precision in fixed tubes. The full proof of the facts found by discovery, as outlined up to this point, lies in the field of pure science. There are definite laws expressed by algebraic equation governing the flow of fluids in tubes. The matter of fluid flow was first investigated by Poiseville,[13] a French physician, in 1843, and later a law was formulated bearing his name. Poiseville's law of fluid flow has been much refined in the light of modern knowledge, so today we have a definite formula for fluid flow, wherein the diameter of the tube, the length of the tube, the quantity or the volume of flow y equals Pi $pr^4t/8LQ$), wherein the viscosity of the flow is expressed in definite units of force called poise and centipoise, in the metric system.*

The application of Poiseville's law to this problem involves also the equation of straight line flow and Fanning's equation for turbulent flow. The equation is (dp equals 4 $flplu^2/2$ gd). The equation for straight line flow is (dp equals 32 ulu/gd^2).*[14].

The underlying facts and theory to be correlated in this problem is whether a warm or cold solution gives better results when injected into the blood stream, which has a definite viscosity, temperature, volume and rate of flow in any given blood vessel of known size and location. The location is only incidental, in that it indicates the diameter of the vessel for injection, being more or less as experience has shown to be required. Other factors are the flexibility of the tube walls, pulsations due to heart action and muscle activity, bends in the tube, etc. The pulsations are damped out by the elastic walls of blood vessels. When all of these factors have been settled by experiment, then it may be possible to make an injection into the blood, with some degree of certainty as to the outcome.

The physical and chemical factors of the blood have long since been definitely determined and found to be constant for all practical purposes; our attention becomes centered on what the physical and chemical constants of any fluid must be for successful x-ray pictures, after injection into the blood vessel and flowing blood stream having these fixed physical and chemical constants.

The evident temperature effect requires explanation in such manner as to correlate the facts with the underlying theory. The equations of straight line and turbulent flow make apparent the relationship of the different physical factors involved in the flow of any one liquid at any one temperature and the point where straight line flow changes to turbulent flow. The laws of flow show that the greater the viscosity of any liquid the more it tends to straight line flow and vice versa. Straight line flow is understood to mean flow without mixing or rolling in eddy currents such as occurs in turbulent flow.

Since liquid of low viscosity—that is, very free flowing—tends to turbulent flow, any such solution soon spreads itself through a large volume of blood, in this case resulting in such extreme dilution of the opaque medium as will prevent obtaining good x-ray pictures.

The problem becomes more complicated by the fact that two liquid solutions are involved and at two different temperatures. It is evident that to obtain good x-ray pictures straight line flow is required, or in other words more viscous flow, for the reason that extreme dilution of the iodin compound must be prevented up to the point of the obstructive lesion.

The rate of flow for this problem has not been definitely established, but it is a factor in knowing the time required for the opaque

---

μ equals Greek Mu. ρ equals Greek Rho.

medium to reach a certain distance from the point of injection: depending upon the cross sectional area of the blood vessel. The theoretical equation for rate of discharge through an orifice is V equals $C\sqrt{2gh}$. It is well known that a lowering of temperature increases viscosity, and we know that viscosity of the blood remains practically unchanged. We know from the flow laws and formula that any liquid less viscous than the blood will cause turbulent flow, which means rapid mixing of the solutions, consequently more rapid dilution of the solution. The opposite must be the objective desired. Temperature is found to play the more important part in the mixing of two solutions of different viscosity.

One of these solutions, the blood, we know remains at a constant temperature and viscosity, and any solution injected will eventually become the same temperature, followed by more intimate mixing and dilution of the iodin compound. This is desired at a point away from the region of inspection but not at the point of inspection. Since a solution of low temperature accomplishes these results the next answer to be sought is why?

This may best be illustrated by the example of two liquids, say syrup and water, each having different viscosities, as we know. Assume the water is at a fixed temperature of 98° F., at which temperature it has a viscosity coefficient of .690 centipoises. Then assume that the syrup of 40 per cent sugar content also to be at 98° F. temperature and viscosity coefficient of approximately 35 centipoises. The two solutions are now mixed and the time rate of complete mixing observed, which is then repeated with the same solution, except the syrup which has been previously cooled to, say, 40° F. It will be noted that the time required for complete miscibility to occur is much longer.

With these same solutions and physical conditions we next observe what happens in flowing through tubes. We find that the solution or syrup at 40° F. flows more slowly and without turbulence. If followed by water or blood, the center flows faster and the blood takes this center and smaller channel, leaving a layer of more viscous liquid next to the tube wall to be gradually washed along.

However, in the case of injection into the blood vessel a different condition exists. The blood vessels are warm and will soften the layer next to the wall, thus reducing the viscosity in this thin layer, forming a cylinder of lubrication next to the vessel wall, causing the middle stream of more viscous solutions to be pushed ahead as a dense plug by the blood, just as a lubricated piston moves along in a cylinder, and although the method of lubrication is different the

effect is the same. This keeps the blood vessel fully distended and also filled completely with opaque solution. It is readily seen that a peripheral layer of lower viscosity of the injected solution occurs after injection, due to the warm blood vessel walls increasing the temperature in the peripheral layer of injected fluid.

The moving plug persists through the entire region of inspection, but is later churned up with the blood in the heart and eliminated from the body, with no harmful effects to any organs or tissues, nor any functional disturbances to any of the organs or tissues.

The smaller the blood vessel the less viscous the injected solution needs to be, because there is little or no turbulent flow in small tubes. This fact makes possible the use of a warmer solution than is injected into larger vessels, and consequently a smaller needle can be used.

A further detailed study is necessary, of the equation of flow and the point of critical velocity, which is the velocity of flow where turbulent motion begins. The critical point of flow is obtained by equating the equations for straight line flow and turbulent flow for equal pressure drop. It may be assumed that the pressure drop is the same in the blood stream at any one point, therefore it is proper to equate these two equations (dp equals 32 y lu gd^2), and (dp equals y $fplu^2/2gd$).

Therefore equating the Poiseville and Fanning equation, we have (32 y lu/gd^2 equals $2flpu^2$ 2gd), and from this (U) equals 942 y/s d) for all fluids for values in feet and decimal feet.

In this problem the object sought is a greater critical velocity for the injected fluid than that of the blood. The extent of this difference is determined by experiment where the values are selected that produce the best results consistently. The use of the laws and equations of flowing fluids make possible a positive scientific analysis of what is taking place when it is desired to obtain a definite result from intravascular injections for x-ray pictures.

From such experiments data is obtained for finding the definite physical constants of the solution to be injected, which experiment has proven to give the best result.

The entire process can be studied with blood and solution in the laboratory in glass tubes in a glass water bath adjusted to body temperature. The flow of blood can be simulated in the tubes by actually using blood itself. The tubes are so provided that injection

of the opaque fluid or solution can be made similar to a blood vessel injection.

Experiments in rubber tubing can be carried out also. These experiments are now being conducted and will be reported in a separate paper. Withdrawal of blood into a syringe, filled with 50 per cent glucose, prior to intravenous injection, shows the relative viscosity and miscibility.

Diffusion plays no part of consequence in the problem of intravascular injection because it is far too slow a process to effect dilution of the opaque medium to any appreciable amount.

The energy and time required to cause diffusion are too large to be a factor of dilution in the time devoted to making x-ray pictures as has been described.

The study of injections into the fixed tissues, such as muscles, brain cavity and other organs, is in progress. Such injections appear practical. The lymph stream is the vehicle that carries the solution to the desired location in the tissues. This is a problem to be dealt with mathematically in the field of infiltration or convection.

CLINICAL APPLICATION.

The use of this medium for intravascular studies has rather unlimited possibilities.

From previous description of the manner of the physiodynamics of the problem we are dependent on the circulating blood to transport the solution; therefore it must always be injected distal to the point of the suspected vascular lesion. In the present stage of development it is not so well adapted to arterial studies as to venous studies, but fortunately our clinical problems are more often confind to the venous system. It is distinctly more applicable for the study of lesions within the venous system than in the arterial.

For the study of intracranial venous lesions the principal channels that are involved as those lying adjacent to the cranial vault and are readily accessible to direct injection. The ideal medium would be one that could be injected into the arterial supply, i. e., the carotid artery, carried to the brain, and the shadows studied upon their return through the venous system. Obviously this calls for a large amount of solution, which on account of the overlapping of the vascular shadows may produce a confusing picture. This has already been tried with only fair success. In the dog, which has a much smaller brain in comparison with the human, injections into the carotid artery have

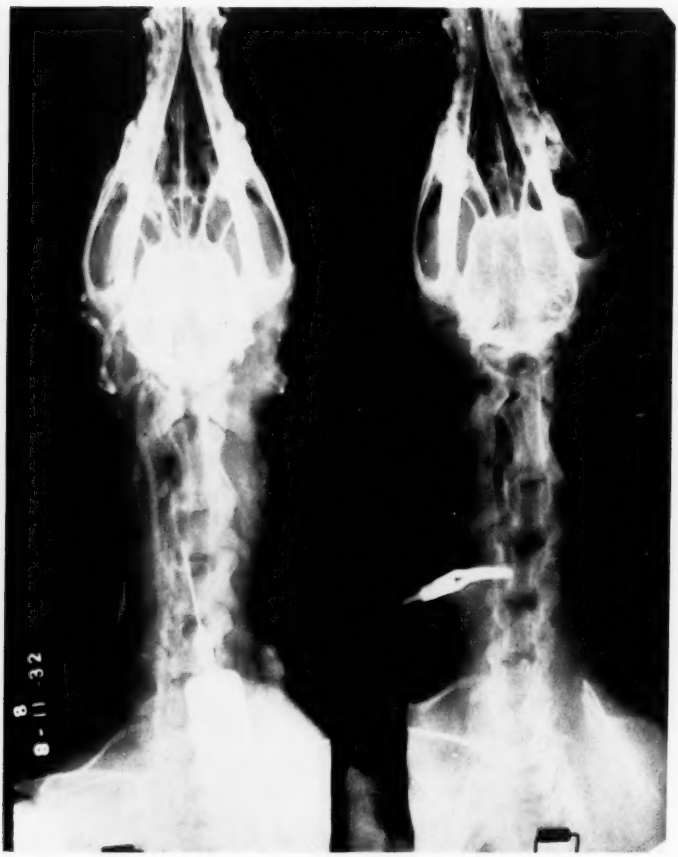


Fig. 2a. This picture shows the shadow, in the right internal jugular vein, of the returning solution that was being injected into the right carotid artery. The external jugular vein had previously been obstructed. The brain shows a distinct opacity, but no vascular detail can be made out.

Fig. 2b. This shows the same animal with the picture taken six minutes later. The brain cavity is clear and there are no remaining shadows in the veins.

been made. The larger arterial vessels can be identified as well as the returning venous flow, yet the medium is so diluted on its return that the contrasts are not always satisfactory. (Figs. 2a and 2b.)

It may be desirable to study all of the intracranial dural sinuses, by one injection, as illustrated by the following case:

E. H., a forty-year-old man, on October 22, 1934, was admitted to the hospital with a large carbuncle on the back of the neck, which after five days was radically removed. Two days following this operation he had a slight chill and convulsion and immediately following he collapsed into unconsciousness from which he did not recover. He developed a flaccid paralysis of his right arm; his temperature varied from 101 to 102.5° F., his pulse rate was in the 80's, his respiration in the low 20's, with a leucocyte count of 16,400, and his spinal fluid was normal with an increase in pressure.

Dr. Frank Teachenor of Kansas City, who had called me to make these radiographic studies, made a buttonhole trephine over the longitudinal sinus near the junction of the frontal temporal bone. Through this opening I injected the radiopaque solution. In the first series the concentration was not great enough to give a distinct shadow, but later, by chilling and increasing the viscosity and the concentration of the solution, very satisfactory shadows were procured, which definitely ruled out any blocking in the intracranial sinuses, as the shadow could be followed from the point of injection down to the edge of the plate into the internal jugular vein. (Fig. 3.) An accident occurred during one of these injections (Fig. 4) which emphasized the value of a nonirritating and quickly absorbable solution. The point of the needle pierced the floor of the exposed longitudinal sinus, which permitted the opaque solution to leak out between the two hemispheres of the brain. There was no reaction by the patient and the accident was discovered only when the film was developed. As soon as additional films could be taken, which was about twelve minutes later, this shadow had completely disappeared. Finally, after this man had died of an extensive encephalomalacia of the left frontal lobe, which would have eventually developed into a brain abscess, could he have survived long enough, injections of the opaque solution were made into each sigmoid sinus, which brought out certain points in the x-ray technic.

One distortion (Fig. 1), which apparently shows the internal jugular vein, going down the back of the neck, was caused by the improper position of the head and neck at the time the picture was made. Another picture illustrated the possibility of using this me-

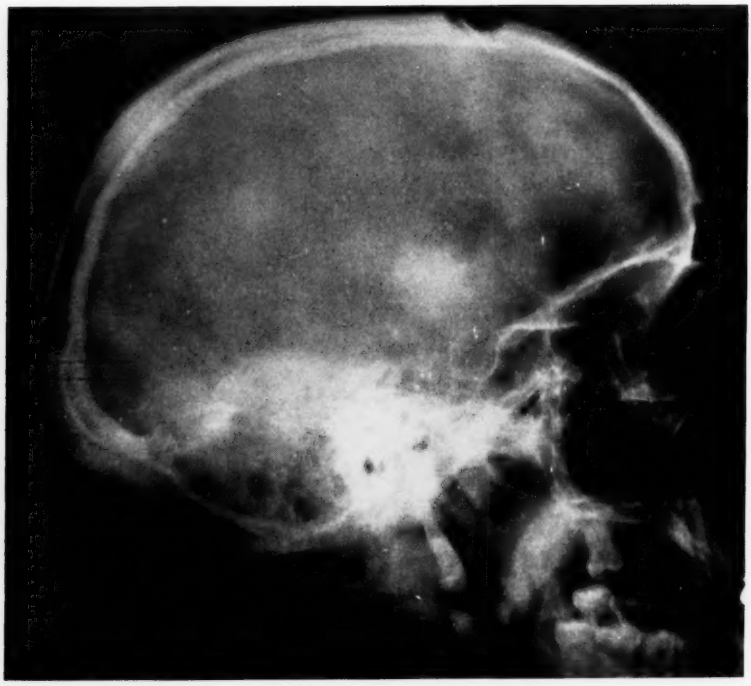


Fig. 3. A complete shadow of the longitudinal sinus from the point of injection at the site of decompression, including the jugular bulb and the internal jugular vein. This demonstrates that the vessel is patent throughout.

dium in the postmortem studies of the anatomy of certain vascular areas. The thorax had not been opened, and the lower end of the jugular vein was still blocked by postmortem clots when this solution was injected under moderate pressure. There was a retrograde extension into the tributaries, which apparently extended into the pterygoid and pharyngeal plexus. While this is principally of academic interest, it suggests the possibility of using this or some other radiopaque medium for postmortem studies, where necropsy permits cannot be obtained or where it is undesirable to disturb certain vascular areas. It may be also used as a matter of permanent record to illustrate the vascular lesion.

When the suspected lesion is quite distant to the point of injection it is obviously necessary to use a sufficient quantity of the solution that would carry beyond the site of the suspected lesion, otherwise confusion may result as to the interpretation of the termination of the shadow, as it could not be determined whether this shadow was due to actual blocking of the vessel or the normal limits of the intravenous opaque medium. This again emphasizes the value of glucose as a vehicle. In the dog, I have been able to determine accurately, lesions along the course of the inferior vena cava by the injection of this medium into the saphenous vein. While I have not yet had the opportunity to make the same observation in the human, I see no reason to believe that it would not be quite satisfactory. As it is nonirritating, inert and readily absorbed, it should also be an ideal medium for the study of suspected spinal cord lesions. I have used it in the study of sinus tracts. Because it is absorbed so readily by the large surface of granulation tissue, the x-ray picture should be made immediately after the injection. Again, it has this advantage, that we need not be concerned about any local irritation or the retention of any foreign material which might be confusing when additional x-rays are made.

For the diagnosis localization and accurate determination of the boundaries of other vascular lesions, such as arteriovenous aneurysms, it is distinctly applicable. It should be of distinct service in the differential diagnosis of thrombophlebitis of the extremities. Not only are its inert properties advantageous, but it can be injected remotely away from the site of the acute inflammatory reaction. I propose to use it also in the differential diagnosis of selected cases of retrobulbar abscesses and cavernous sinus thrombosis, keeping in mind the danger of traumatizing the angular vein.

The following cases illustrate some of the clinical applications of this problem:

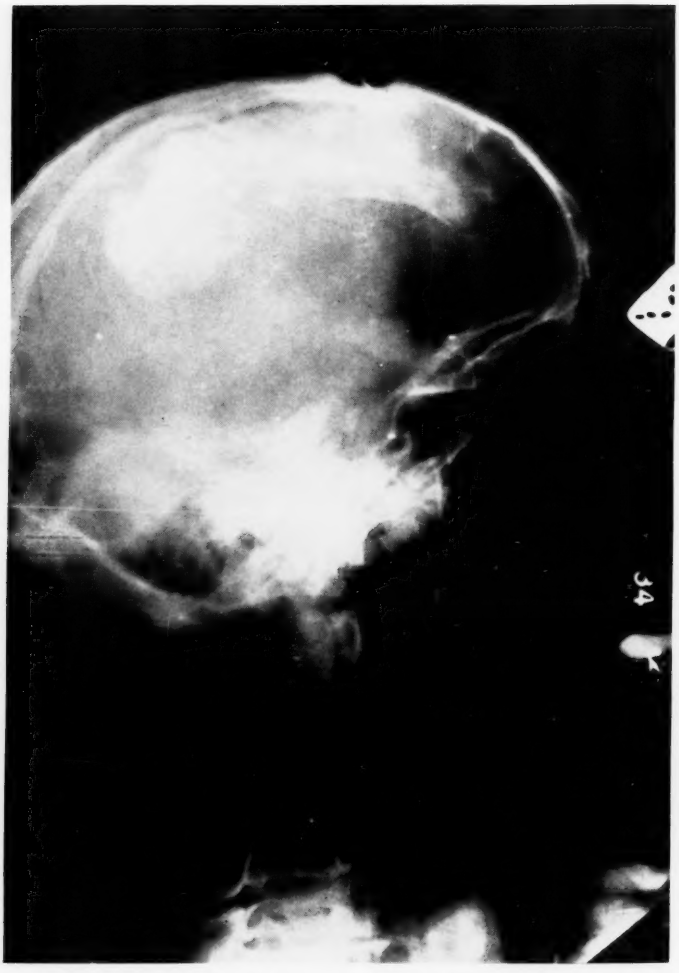


Fig. 4. Shadow produced by the accidental injection of this opaque solution through an opening made by the point of the needle in the floor of the longitudinal sinus at the site of injection. As soon as another picture could be taken, which was about ten minutes after the injection, this shadow had completely disappeared.

REPORT OF CASES.

CASE 1.—D. R., a nine year old boy, on June 12, 1934, four days following a bilateral mastoidectomy, with the exposure of both sinuses, had a hard chill followed by a sharp rise in temperature. His blood culture was positive. A swollen right elbow indicated a metastatic involvement. Each sigmoid sinus was injected with the solution, and the obstructive lesion was found by x-ray to be in the vessel, which had the smooth normal appearing wall.

While the last injection was being made, a sudden warning to avoid the uncovered overhead wires, caused me to accidentally pass the point of the needle through the floor of the sinus and inject some of the solution beneath the dura. (Fig. 5.) The entire procedure was done without any anesthesia and the patient showed no reaction to this accident and has completely recovered from the injection. The x-ray pictures taken soon after the accident, failed to reveal any evidence of a remaining shadow. This accident illustrates the necessity of using a solution, which possesses all possible safety factors.

CASE 2.—S. F., a six year old girl, upon whom I had removed an infected thrombus from the sigmoid sinus, in 1932, and repaired with a viable muscle flap, I elected to use this opaque solution, to prove that the sigmoid sinus had recanalized. Without any anaesthetic, this child was placed on the x-ray table, and a lateral x-ray was made of the head and neck for a control. This opaque solution was then drawn into a five cc. glass syringe, and with a 26 gauge needle, two cc. were injected through the skin into the sigmoid sinus. As the concentration of the opaque medium was very low, only a faint shadow was seen, but there was enough solution passed along to definitely establish the patency of the vessel. The patient was not disturbed and immediately left the hospital after this examination.

CASE 3.—N. T., a five year old girl, who was convalescing from measles and had a bilateral suppurative otitis media, suddenly had a hard chill. Her temperature rose to 104°, with a 26,000 leucocyte count. The chill was repeated twenty-four hours later. Under ether anesthesia a mastoidectomy was performed and the sigmoid sinus was exposed. The iodine compound and glucose was injected, but the x-ray showed a blocking near the bulbar end, which also was indicated by the shadow of the solution in the deep posterior occipital vein. Three injections were made on this child, and in no way was she disturbed by the procedure.

CASE 4.—R. T., a 14 year old boy, was admitted to the hospital, March 24, 1934, and for the past year I have had the distressing experience of attempting to establish a better drainage from a brain abscess of cranial osteomyeletic origin. Injections of this opaque media into the fistulous track, have established leads, which when followed, opened into new abscess cavities. At no time has the patient been disturbed by these injections, which have been made with only the use of a small amount of gravity pressure. Care should always be taken to avoid the rupture of the pyogenic capsule, enclosing the brain abscesses. If some of the solution escapes upon the scalp, confusing shadows will result. Where a definite abscess is found and pus is aspirated, if the shape, size, and location are desired, the solution may be injected in the amount of one-half of that of the pus, which has been removed.

The rapidity of the absorption of this solution and its prompt disappearance from the site of the injection is well illustrated by the following case:

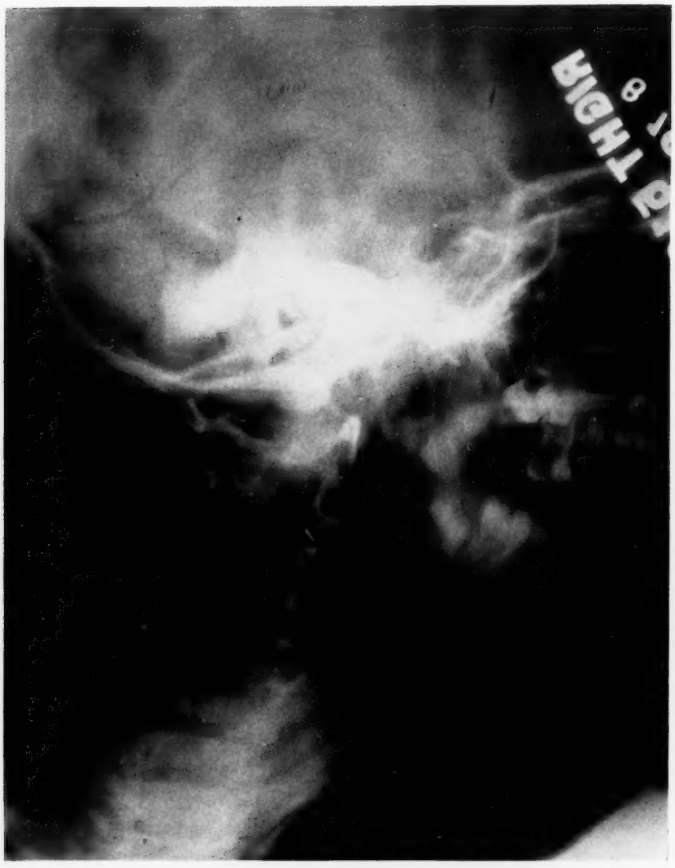


Fig. 5. Shadow produced by the accidental escape of the opaque medium beneath the dura, while injecting the sigmoid sinus. This in no way disturbed the patient and was quickly absorbed.

CASE 5.—A 26 year old man with a draining fistula over the anterior surface of the middle third of the leg, of two years' duration, was injected with four cc. of a warm opaque solution. The solution could be felt beneath the skin, as it entered the subcutaneous granular tissues.

The roentgenologist did not know that the injection was being made and was not ready to take the picture until about four or five minutes later. By that time the solution had so disappeared that only a faint shadow was obtained. The relative opacity of the solution is well illustrated by the droplets that can be obtained within the metal needle on the syringe, and it is well to always show some of the solution outside of the body on the plate, in order to determine the relative opacity.

SAFETY FACTORS.

The opaque media, which I have described for the study of vascular lesions by x-ray seems to possess all the necessary safety factors, as it is inert, nontoxic, does not produce embolic phenomenon, is nonirritating to the vascular endothelium, is quickly and completely eliminated and, even when accidentally or intentionally injected outside of the vascular system, such as the subdural space, it is without harmful effects. There is one factor, however, which must be constantly kept in mind—that is, that the patient may have an allergic hypertonic susceptibility to any form of iodin. Proper tests should be made to determine this factor prior to injection. Although this iodin is firmly bound and apparently inert, it might react badly in these allergic patients. Glucose seems to be a safe and proper vehicle, and lowering the temperature of the injected solution from 20 to 30 degrees F. below normal body temperature apparently produces no harmful effects to the patient. Finally this paramount factor in the use of any therapeutic or diagnostic procedure must always be for the safety of the patient.

Dr. Galen Tice, of the University of Kansas School of Medicine, and Mr. Mark Carroll, my technical assistant, have been very generous in their help with this problem.

PROFESSIONAL BUILDING.

BIBLIOGRAPHY.

1. Dixon, O. Jason: Experimental Studies in Vascular Repair. J. A. M. A., 103:1129-1134., October 13, 1934.

1a. Dixon, O. Jason: Research Studies in Visualization of the Vascular Supply of the Head and Neck. Trans. Am. Acad. Oph. and Otolaryng., 1934.

1b. Trendelenburg: Schussverletzung des Herzens mit Einheilung der Engel. 31 Kongress des deutschen Gesselschaft fur Chirurgie, 1902.

2. Reithus, O.: Ueber einen Fall von Schussverletzung des Herzens mit Eiheilung des Projectils nebst experimentellen Untersuchungen uber Fremdkorper im Herzen. Deutsch. Ztschr. f. Chir., 67:414, 1903.

3. Franck and Alwens: Kreislaufstudien am Rontgenschirm. Munchen. Med. Wchnschr., 57[1]:950, 1910.

4. Scheppelmann: Uber Blutgefasse atten im Rontgenbilde. Munchen. Med. Wchnschr., 57[2]:1614, 1910.

5. Berberich, J., ans Hirsch, S.: Die Rontgenohraphische Darstellung der Arterrie U. Venen am Lebenden Menschen. Klin. Wchnschr., 49:2226, 1933.

6. Brooks: New England Journal of Medicine, 209:26, December 28, 1933.

7. Dos Santos, R.: L'arteriographie des membres. Bull. et mem. de la Soc. nat. de chir., 58:635, 1932.

8. Radt, P.: Eine neue Methods Zur Rontgenologischen Sichtharmachung von Leber und Milz durch injektion eines Kontrastmittels. Med. Klin., 26:1888, 1930.

9. Thorotrast: Roentgenologic Demonstration of the Parenchyma of the Spleen and Liver by Means of a New Intravenous Contrast Medium (Thorotrast). Dr. Silvije Kadrnka, Geneva, Switz.

10. Allen, E. V., and Camp, J. D.: Roentgenography of the Arteries of the Extremities. Proc. Staff Meeting of the Mayo Clinic, 46:657, 1932.

11. Frenckner: Acta Oto-Laryngologica. 20:10134, 1934.

12. Edwards, Edw. A.: The New England Journal of Medicine. 209:26, December 28, 1933.

13. Poiseville: College of Physics. Reed and Guthe, 1912.

14. The equations and viscosity material in this paper are taken from the following sources:

1. The Principles of Chemical Engineering. Walker, Lewis, and McAdams, pages 75-100.
2. One Hundred and Forty-five Tested Solutions. Chemical and Metallurgical Engineering. McGraw, Hill, page 174.
3. Gen. Chemistry and Biophysics. Steel, pages 74-295

XLI.

IMPERMEABLE CICATRICIAL STRICTURE OF THE ESOPHAGUS TREATED BY A MODIFICATION OF THE IGLAUER TECHNIC.

HERMAN J. MOERSCH, M. D.,

ROCHESTER, MINN.

The treatment of impermeable cicatricial stricture of the esophagus constitutes a very serious endoscopic and surgical problem. Fortunately it is not a common complication. It is, however, one that must be carefully guarded against in every case of benign cicatricial stenosis of the esophagus. It is most likely to develop as a result of neglect, but it may develop as the result of inadequate treatment. One of the most common therapeutic errors is the tendency to regard gastrostomy as the first and only procedure in the treatment of benign cicatricial stricture of the esophagus, disregarding the necessity of maintaining a patent esophageal lumen.

Among 310 patients with benign cicatricial stricture of the esophagus who were seen at The Mayo Clinic, there were seventeen who had impermeable stenosis. Heindl found this complication six times in 137 cases of cicatricial stricture of the esophagus seen at the Vienna Clinic. This would place the incidence of the complication in cases of benign cicatricial stricture of the esophagus at about five per cent.

Patients with an impermeable cicatricial stricture of the esophagus almost invariably have a gastric stoma at the time they present themselves for treatment. While they are usually able to maintain their nutritional requirements by this means, they are generally very unhappy and miserable, and they will gladly undergo a serious surgical or endoscopic procedure to do away with such a gastric stoma.

Great care must be exercised before establishing a diagnosis of impermeable cicatricial stenosis. Cicatricial strictures of the esophagus are notorious for their ability to close completely for a variable period of time and again reopen spontaneously, even so long as a week or more after closure. It is indeed embarrassing to undertake a serious surgical or endoscopic procedure and later find the esophagus has reopened of its own accord. When complete esophageal ob-

struction occurs in a case in which the patient has not undergone a gastrostomy, before opening the stomach for feeding, the patient should be hospitalized and immediately given 10 per cent glucose and 1 per cent saline solution intravenously for at least a period of a day or two. This serves a twofold purpose: it overcomes dehydration and makes the patient a less serious operative risk. More important, it frequently causes an apparently impervious esophageal lumen to open sufficiently to permit liquid to pass through into the stomach. If this occurs, the patient should promptly be started swallowing a fine, twisted silk thread. This thread will invariably pass through the stricture, as it will follow anywhere water will pass. Once the thread has passed beyond the stricture and become fixed in the intestinal coils, the stricture can rapidly and safely be dilated from above, the patient being spared gastrostomy. The habit of attaching a small shot to the end of the thread to facilitate deglutition is to be avoided, as it often defeats its own purpose by plugging whatever lumen is present. The above procedure will at times be found of value even in cases in which patients have a gastric stoma.

Esophagoscopy is indicated in every case of complete esophageal obstruction, and especially so if it is sudden in onset. A foreign body may be found completely obstructing an otherwise patent cicatrical stricture, and its removal may afford relief. Esophagoscopy likewise enables one to determine better the character of the stricture and the possibility of its dilatation and separation by passage of filiform bougies. If the patient has a gastric stoma, it is often advisable and useful to examine the stricture from below by way of this stoma, as one may be able to open the stricture more readily from below than above, as has been demonstrated by Iglauer.

The patient should also be examined fluoroscopically to determine the permeability of the stricture and its length. Lipiodol will be found of especial value as the contrast medium. In the presence of a gastric stoma one fails to find a trace of any chemical substance, such as methylene blue, in the stomach when it is introduced from above.

A diagnosis of impermeable cicatricial stricture of the esophagus having been established, the patient having a gastric stoma, there remain various available procedures to re-establish a continuous passageway to the stomach. Such procedures may be divided into two chief groups: surgical and endoscopic. The type of operation selected must necessarily depend on the type of obstruction, its situation and extent, and on the surgeon's experience. When feasible, however, the endoscopic procedure should be given preference, as it is less

drastic, takes less time, and if successful leaves the patient with a more serviceable and satisfactory apparatus for deglutition. Many different endoscopic procedures have been described and advocated in the treatment of impermeable cicatricial strictures of the esophagus. King has given an excellent review of the literature pertaining to the subject, adding a successful procedure of his own.

Recently a patient with an impermeable cicatricial stricture of the esophagus was operated on at the clinic by a modification of the technic devised by Iglauer. This procedure was so satisfactory and easy of performance in this case that it was thought worthy to report it:

REPORT OF CASE.

CASE 1.—The patient was a man twenty-three years of age, who came to the clinic in December, 1934, complaining of complete esophageal obstruction. This had resulted from cicatricial stricture of the esophagus secondary to a caustic burn two years previously. He had been unable to get anything down by mouth for at least a week before coming to the clinic, and on attempting to swallow liquids, they were regurgitated immediately. He likewise was constantly expectorating saliva.

The patient was hospitalized, and 10 per cent glucose and 1 per cent saline solution were administered intravenously in amounts of 1000 cc. twice daily. While this overcame dehydration, it did not relax the esophageal obstruction, and he was unsuccessful in swallowing a thread. Esophagoscopy was performed two days later and a dense stricture was found involving the lower portion of the esophagus. There was a very small dimpled area in the center of the stricture, with a surrounding area of granulation. An attempt was made to pass a filiform bougie through the stricture, but it was unsuccessful as was also an attempt to pass an olive-tipped wire. Forceps were then inserted into the dimpled area and the stricture was dilated. While some progress was made in separating the stricture, it remained impermeable to the passage of air or water forced into it. Fluoroscopic examination likewise failed to reveal a passage through the stricture. Intravenous administration of glucose was continued two days longer at the request of the patient before gastrostomy was undertaken. It was decided at the time of gastrostomy to try a modification of the Iglauer cystoscopic technic for the treatment of impermeable cicatricial strictures. Instead of passing a ureteral catheter into the esophagus from below by means of a cystoscope through the gastric stoma, it was introduced from above. After the stomach was opened, the esophagoscope was introduced and a small caliber, hollow brass tube, through which an olive-tipped, number 8, leaded catheter had been threaded (Fig. 1), was carried down to the stricture and introduced into the small area opened previously with forceps. The catheter was then gently pushed through the brass tube and into the stricture. Gentle pressure was made from below through the open stomach. The catheter was gradually forced into the stomach, where it was grasped by the operating surgeon, Dr. C. W. Mayo. A thread was then attached to the catheter and was withdrawn through the stricture and out of the mouth, making a continuous circle and a suitable means to carry on future dilatations. The leaded catheter and hollow brass tube were used as they

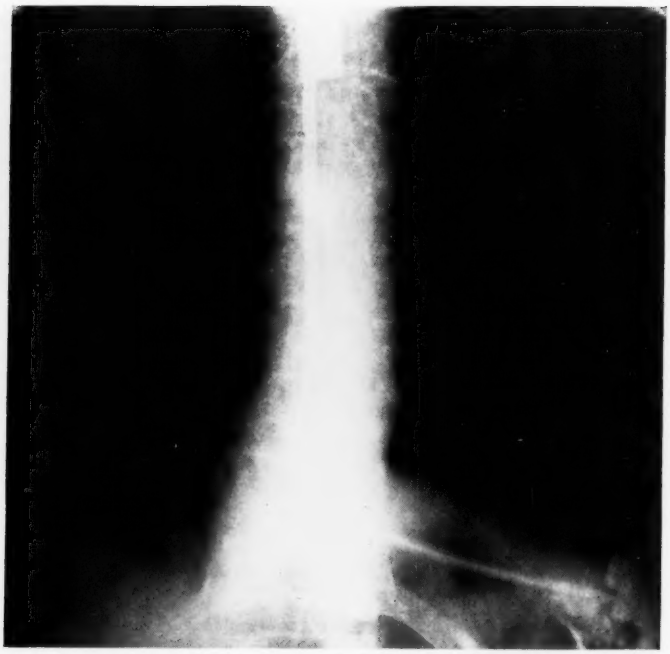


Fig. 1. Roentgenogram showing method of passing the catheter through the tube in the esophagoscope.

gave more body to the catheter so that pressure could more readily be applied. It likewise has the advantage that the catheter readily follows the course of least resistance and is sufficiently pliable that enough pressure could hardly be applied to perforate the esophagus. While this procedure would probably not be applicable in all cases of impermeable cicatricial stricture of the esophagus, it was found eminently successful in this case. It offers another adjunct in the treatment of impermeable cicatricial stricture and it should be applied in conjunction with Iglauer's technic before attempting the more serious operative procedures.

BIBLIOGRAPHY.

Heindl, Adalbert, Jr.: Klinische Beobachtungen an 137 gutartigen Osophagusstenosen der I. chir. Universitätsklinik Wien 1901-1925. Deutsch. Ztschr. f. Chir. 199:252-269, (Nov.) 1926.

Iglauer, Samuel: Impermeable Stricture of the Esophagus Relieved by Retrograde Bouginage with the Aid of a Cystoscope Inserted Through a Gastrostomy. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:1191-1202 (Dec.) 1932.

King, Edward: A New Method of Restoring the Lumen in a Case of Impermeable Stricture of the Esophagus. Surg., Gynec. and Obst. 39:596-598, (Nov.) 1924.

XLII.

BIOCHEMISTRY RELATIVE TO OTOLARYNGOLOGY.*

R. N. HARGER, PH. D.,

INDIANAPOLIS.

The subject of my remarks was suggested to me by the late Dr. John W. Carmack, who was my good friend as well as your good friend. I told Dr. Carmack the while biochemistry has made notable contributions to many fields of medicine I was not sure that it has a great deal of a constructive nature to offer to those interested in otolaryngology. I suggested that I might perhaps discuss some modern fallacies which involve biochemistry as well as otolaryngology. I am simply a biochemist and not a physician, much less a specialist in your fields, and it will of course be understood that any clinical applications of my talk will be taken with a liberal supply of sodium chloride.

Biochemists have been interested for a long time in the function of the respiratory tract involving the transportation of gases. We know that this tube serves both as intake and exhaust for the body engine and that during a period of twenty-four hours it carries about 400 cubic feet of air and furnishes the body with from one to four pounds of oxygen and carries away from the body one and one-half to six pounds of carbon dioxide and something like a pound of water. Since the air cleaning arrangement of this system is not any too efficient it is not surprising that dust particles and micro-organisms are deposited along this tube and that these foreign bodies sometimes give rise to infection.

The first subject I wish to discuss is the relation between vitamin A and infection of the upper respiratory tract. Vitamin A has been called the "anti-infective vitamin." I believe that this term was introduced by Mellanby in 1928.[1] This term is widely used these days, particularly in advertisements for the laity appearing in drug stores, broadcast over the radio, and seen to a certain extent in medical journals. Our knowledge regarding vitamin A is based upon fundamental research in biochemistry and the allied sciences, this

*Address delivered before a meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society, Inc., at Indianapolis, Indiana, on January 23, 1935.

subject having occupied the attention of several brilliant investigators. As a result of this work we know the distribution of this vitamin in various foods and medicines and also a good deal regarding its chemical nature. We know that it is related to carotene in that the animal is able to absorb carotene from the intestinal canal and to convert this carotene into vitamin A, which is stored in the body, particularly in the liver. Now carotene is nothing but the yellow pigment appearing in carrots, sweet potatoes, butter, etc., and it has been known to biochemists for many years, but its relationship to vitamin A was discovered only recently. When rats are placed upon a diet which is deficient in vitamin A they will develop a condition in which the mucous membranes exhibit keratinization, which is a pathologic deposition of horny epithelial tissue. As a result, such mucus surfaces are more easily invaded by micro-organisms. In this condition there is no question that rats and some other animals are more susceptible to infection, especially of the upper respiratory tract. On the other hand, it does not follow that animals which are not suffering from this condition will be protected from infection of the upper respiratory tract by the administration of excess vitamin A. As a matter of fact, the normal diet contains rather large quantities of vitamin A. Thus a quart of milk will contain about as much vitamin A as a teaspoonful of U. S. P. cod liver oil, and such foods as carrots or spinach are about one-tenth as potent in vitamin A as is cod liver oil. We hear much over the radio about certain well known cough drops, which are said to protect against the common cold because they now contain carotene or "primary vitamin A." As a matter of fact, the use of the word "primary" is a sort of misrepresentation because carotene is not vitamin A but a precursor of vitamin A. The label on these cough drops states that one *box* of this patent medicine contains "700 U. S. P. X. (1934 Rev.) Units of Vitamin A in its vegetable form (Carotene)." This sounds quite impressive until you figure up just how much vitamin A this represents, when you find that this amount of vitamin A would be present in about one-half ounce of fresh carrots or one-half pint of milk!

It would seem to be just about as logical to call insulin the anti-infective hormone as to claim that vitamin A is the anti-infective vitamin, because diabetics are notoriously prone to infection. However, no one claims that a normal individual's resistance to infection will be raised by excessive doses of insulin. Now, just because some animals will develop infection when placed on an artificial diet deficient in vitamin A it does not necessarily follow that these same animals or human beings receiving a normal diet will be benefited in the slightest by increasing their vitamin A intake.

The only way to settle this question is to administer vitamin A to a large number of human beings and see if their resistance to respiratory infection is any greater than that of a similar number of controls. This has now been done by a number of investigators. Wright, Frost, Puchel and Lawrence gave a number of infants halibut liver oil and compared their resistance to infection with a similar group of infants used as controls. They could observe no difference in the two groups.[2] Barenberg and Lewis, in 1932,[3] compared cod liver oil plus viosterol with an ordinary diet. They used some 60 to 113 infants in each group and found no difference in the incidence or severity of respiratory infections in these two groups. In 1933 Hess, Lewis, and Barenberg[4] made further studies along this line, using three groups, each containing forty infants. The first group received haliver oil plus viosterol, the haliver oil furnishing 20,000 units of vitamin A per day. The second group received the same amount of vitamin A in the form of carotene and also viosterol. The third group received only viosterol which contains no vitamin A. Each group was given the stock hospital diet which, like most normal diets, contained a fairly liberal amount of vitamin A. The three groups showed almost identical behavior as regards respiratory infections. In the same year Clausen at Rochester published a paper[5] dealing with the histories of some 1300 children, part of whom had received liberal therapeutic doses of vitamin A. Here again the results with vitamin A were negative. Clausen also made analyses for vitamin A content on the livers of seventy children coming to autopsy. His results showed that these livers contained rather large stores of this vitamin. The alleged protective action of vitamin A for adults was studied by Shibley and Spies[6] at Western Reserve University, using college students and nurses. The subjects were divided by lot into three groups, each group numbering about seventy. The first group received a weekly dose of 200,000 units of vitamin A and 4,000 units of vitamin D. A second group received a weekly dose of viosterol, giving 4,000 units of vitamin D and the third group served as controls. This experiment lasted over a period of one year. The incidence of colds in the three groups was almost identical, and the three curves showing the monthly incidence of colds could practically be superimposed upon each other, including all peaks and depressions. These workers feel that their results furnish "suggestive but not conclusive evidence that vitamin A shortens colds slightly in the winter months." They point out, however, that viosterol alone also seemed to lower the average duration of colds by one or two days so that the effect of halibut liver oil might be ascribed to its vitamin D content. The only recent paper which tends

to support the claim that respiratory infection may be prevented or shortened by vitamin A is the work of Gardner and Gardner[7] in California. Their control group showed very little more respiratory infection than the group receiving vitamin A, which result they explain by saying that the groups were not fairly selected and that the control group contained a larger proportion of resistant individuals.

These results certainly do not lend much support to the claim that vitamin A is the anti-infective vitamin. Please do not understand me to say that therapeutic doses of vitamin A may not be beneficial in some cases. All I am trying to point out is that the available evidence does not furnish much justification for the present widespread use of vitamin A as a panacea for upper respiratory infection.

As regards the other vitamins, our present diet is quite rich in vitamin C, due to the widespread use of fresh fruits and vegetables in addition to such articles as canned tomatoes, etc. My medical colleagues tell me that scurvy is indeed rare these days. In the case of vitamin D there seems to be some justification for enriching certain foods in this factor. The vitamin D content of milk ranges from about 40 U. S. P. units per quart in the summer to almost zero during the winter. At best a quart of milk contains the vitamin D equivalent of about one-half gram of cod liver oil. Most other common foods contain even less vitamin D. For this reason and also because rickets is still too common, the Committee on Foods of the American Medical Association has approved a number of milks which have been enriched in their vitamin D content. The situation here is somewhat confused, however, because the source of the vitamin D is of considerable importance. Clinical tests[8] seem to indicate that it takes from five to ten units of vitamin D in the form of viosterol to equal one unit of vitamin D in the form of cod liver oil and that one unit of vitamin D in irradiated milk is probably equal to at least two units in the form of cod liver oil. Recent work[9] makes it appear that there is more than one chemical substance which has an antirachitic value.

Among other "cures" for the common cold, do you remember the wave of chlorine therapy which swept over this country about ten years ago? This remedy was a product emanating from the Chemical Warfare Service. It survived only a short time and has almost been forgotten. In this connection let me point out that the Chemical Warfare Service has recently reversed itself as to whether permanent respiratory damage can result from gassing with chlorine.

They formerly said that if the victim survived the immediate effects of inhaling chlorine his ultimate recovery would be complete. That this is not correct is indicated by a recent paper by Gilchrist[10] of the Chemical Warfare Service in which a study of a large number of men who were severely gassed in the war indicated that about eight per cent of these men had suffered permanent respiratory damage, which was particularly manifested in bronchitis and bronchiectasis. Chlorine is not the only industrial gas which can cause respiratory damage. Another one is sulphur dioxide. Rostoski and Crecelius,[11] in Germany, recently reported a study of ten cases exhibiting permanent shortness of breath, laryngitis, bronchitis and bronchiectasis as a result of inhaling sulphur dioxide fumes following an accident in a sulfite paper factory. Rostoski and Crecelius first thought that these cases were malingering, but autopsy results in three of the cases showed that definite damage had been done. Another dangerous gas is nitric oxide, which caused most of the deaths in the Cleveland Clinic fire. Here the damage is in the alveoli.

Finally I want to say something about alkalinization and upper respiratory infection. Alkali therapy is dinned into our ears *ad nauseam* over the radio, in the drug store and in alluring advertisements appearing in the press. From a biochemical standpoint the administration of the amounts of alkali usually contained in these medicines would hardly seem to have very much effect upon the body. In the first place, the pH of the body, which simply means the amount of acidity or alkalinity, is one of the body constants which may be compared to the temperature, the blood sugar, etc. If this pH is disturbed, even very slightly, by the administration of acids or bases, the body will at once fight with great vigor to return to the normal value. When alkalis are administered the body will immediately begin to throw alkali overboard by means of the kidneys.

If you wish to test this out, simply swallow a few grains of bicarbonate and note how soon your urine becomes alkalin to litmus. The body fluids of an adult contain about six ounces of alkali, which is mostly sodium bicarbonate. This we call the alkali reserve, and this quantity is maintained at a pretty constant figure in the normal individual. One ounce equals 480 grains, and you can figure for yourself how ridiculous it is to try to alkalinize the body by the administration of a few grains of bicarbonate. It is sometimes pretty hard to interfere with nature. It is, of course, true that you can change the body pH by administering enormous doses of bicarbonate, but this has been shown to be a very dangerous procedure,

and Myers,[12] Orr[13] and others have pointed out that the excessive administration of alkali in the Sippy treatment of gastric ulcer has frequently resulted in alarming symptoms and even fatalities because the body is not able to withstand much of a shift on the alkalin side of its normal pH. The medical literature does not contain a great deal of information relative to the use of sodium bicarbonate in the common cold, but recent results by Diehl of the University of Minnesota[14] indicated that the administration of sodium bicarbonate gave almost identical results as were obtained by the control subjects who received "dummy" tablets containing lactose.

A study by Hilding[15] on nasal secretion during the common cold indicated that as the cold progressed the nasal secretion actually became more alkalin than normal but that during the purulent stage the secretion became more acid than normal. A later paper by Hilding,[16] however, indicates that this change in the pH of the nasal secretion is largely due to the loss or retention of dissolved carbon dioxide in this secretion, depending on the efficiency of aeration of this fluid, and that these results do not indicate any marked acidosis in connection with the common cold.

Of course acidosis may accompany almost any disease which is associated with partial or complete starvation. This is due to the fact that complete combustion of fats requires the burning of some carbohydrates and when inanition persists long enough to deplete the store of liver glycogen the remaining fats will be incompletely burned and acidosis will occur. It does not follow, however, that the administration of alkalies is indicated here, and scientific evidence and modern clinical practice would indicate that a much more rational procedure would be the administration of glucose either by mouth, per rectum or intravenously.

I am sure that in the future biochemistry will furnish information which will prove of value in your fields as it has already done in many fields of medicine.

DEPARTMENT OF BIOCHEMISTRY AND TOXICOLOGY, INDIANA UNIVERSITY SCHOOL OF MEDICINE.

BIBLIOGRAPHY.

1. Green, H. N., and Mellanby, E.: Vitamin A as an Anti-infective Agent. Brit. M. J., 2:691 (1928).

2. Wright, H. P.; Frost, J. B.; Puchet, F., and Lawrence, M. R.: Vitamin "A" and the Common Cold. Canad. M. A. J., 25:412 (1931).

3. Barenberg, L. H., and Lewis, J. M.: Relationship of Vitamin A to Respiratory Infection in Infants. J. A. M. A., 98:199 (1932).

4. Hess, A. F., Lewis, J. M., and Barenberg, L. H.: Does Our Dietary Require Vitamin A Supplement? J. A. M. A., 101:657 (1933).

5. Clausen, S. W.: Limits of the Anti-infective Value of Provitamin A (Carotene). J. A. M. A., 101:1384 (1933).

6. Shibley, G. E., and Spies, T. D.: The Effect of Vitamin A on the Common Cold. J. A. M. A., 103:2021 (1934).

7. Gardner, E. L., and Gardner, F. W.: Vitamin A and Colds. Am. J. Dis. Child., 47:1261 (1934).

8. Hess, A. F., and Lewis, J. M.: Appraisal of Antirachitics in Terms of Rat and Clinical Units. J. A. M. A., 101:181 (1933).

9. Wadell, J.: The Provitamin D of Cholesterol. I. The Antirachitis Efficacy of Irradiated Cholesterol. J. Biol. Chem., 105:711 (1934).

10. Gilchrist, H. L., and Matz, P. B.: Residual Effects Warfare Gases: Use of Chlorine Gas, with Report of Cases. M. Bull. Vet. Admin., 9:229 (1933).

11. Rostoski, O., and Crecelius: Poisoning with Sulfite Gas. Deutsches Arch. f. klin. Med., 168:107 (1930).

12. Gatewood, W. E., Gaebler, O. H., Muntwyler, E., and Myers, V. C.: Alkalosis in Patients with Peptic Ulcer. Int. Med., 42:79 (1928).

13. Orr, T. G.: The Case Against Sodium Bicarbonate. J. Kans. Med. Soc., 33:205 (1932).

14. Diehl, H. S.: Medicinal Treatment of Common Colds. J. A. M. A., 101: 2042 (1933).

15. Hilding, A.: The Common Cold. Arch. of Otolaryng., 12:133 (1930).

16. Hilding, A.: Note on Some Changes in the Hydrogen Ion Concentration of Nasal Mucus. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:47 (1934).

XLIII.

OBJECTIVE AND SUBJECTIVE TINNITUS AURIUM OF VASCULAR ORIGIN: REPORT OF CASE PRESUMABLY DUE TO CEREBRAL ANEURYSM.*

PIERRE VIOLE, M. D.,

AND

ANDREW A. LOVE, M. D.,

LOS ANGELES.

A study of early literature discloses considerable skepticism as to the possibility of diagnosis of cerebral aneurysm during life. Sands[1] states that "the surrounding brain tissue ordinarily adjusts itself well to the slowly expanding growth; hence, their recognition before rupture is well nigh impossible." In the later literature, clinical studies have been reported by many authors which have established the fact that it is a pathologic condition that lends itself to a correct diagnosis during life.

According to Schmidt,[2] who reports a group of twenty-three cases, the first known description of intracranial aneurysm originated from Biumi in Milan in 1765. Green,[3] in a contribution on this subject, in 1878, states that "Henning calls attention to the fact that brain murmurs were first investigated by Fisher of Boston in 1832."

LOCATION, AGE AND SEX INCIDENCE.

According to Gowers, in an article written in 1893, these aneurysms are most frequently found on the middle cerebral artery, and thereafter with decreasing frequency on the basilar, internal carotid, anterior communicating, vertebral, posterior cerebral and the inferior cerebellar arteries. Von Hofman,[4] in an article in 1894, stated the order to be the middle cerebral, internal carotid, anterior communicating, basilar and the vertebral arteries, and the frequency of aneurysms on the basilar artery as being variously computed.

---

*Presented before the meeting of the Western Section of the American Laryngological, Rhinological and Otological Society, Los Angeles, January 28, 1935.

From the Department of Otolaryngology in the School of Medicine of the University of Southern California.

There also are differences of opinion as to the prevalence of cerebral aneurysm on one side or the other, the greater number having been reported on the left side in those reports of cases reviewed by the writer. Lebert[5] states that aneurysm of the internal carotid artery is believed by him to appear more often on the left side because of the fact that on the left side the carotid artery arises directly from the aorta.

There also have been differences of opinion as to the incidence of cerebral aneurysm in the different sexes, the greater percentage, however, having been reported in women. According to Busse,[6] Wichern[7] and Schmidt, age is no factor in its occurrence, it frequently being found in individuals between the ages of thirty and sixty.

According to Beadles,[8] in an exhaustive study of 555 cases, aneurysm of the internal carotid artery in its course through the sinus cavernosis, caused symptoms in 69 per cent of the cases studied. Symptoms, such as paralysis of the eyes, associated with neuralgia of the ophthalmic nerve, diminished vision and trigeminal symptoms along with tinnitus aurium, are manifested in these cases.

OBJECTIVE TINNITUS AURIUM.

Weil[9] states that there are two distinct types of tinnitus aurium easily distinguishable objectively that are the result of two distinct causative agents. The first type is characterized by a peculiar crackling noise not unlike the sound caused by the snapping of two finger nails together. It is caused by a clonic spasm of the muscles in and around the eustachian tube. The muscle most commonly affected is the tensor veli palati. In the second class the sound perceived, both objectively and subjectively, is always a hum or murmur synchronous with the systolic heart sound, and is not an unusual concomitant of cerebral aneurysm. Kafka[10] describes this objective sound as one of a constant or intermittent nature which the examining physician himself can hear. He reports these sounds as being associated with vertigo at times and often being found in aneurysm of the carotid artery.

Poorten[11] believes that objective noises in the ear are usually the result of an aneurysm. Moos[12] reports a case in which persistent subjective noises existed so as to cause insanity of the patient, which case at autopsy showed the jugular bulb to be tremendously enlarged, and he attributes the tinnitus in this case as being due to the enlargement of the bulbus. Impairment of hearing is usually associated with tinnitus aurium in these cases.

Green in his early contribution to objective and subjective systolic murmurs in the ears states: "Poorten gives a resumé of other cases of objective murmurs which have been described. The first of these was stopped by compression of the arteria auricularis posterior and is referred by Grüber[3] to be an aneurysm of the basilar artery and by Von Troelitsch to peculiarities in the branches of the posterior auricular artery and changes in the parts to which this artery is distributed. In the second case there was a systolic murmur in the heart, ear and head, which probably was a transmitted sound from the heart, and in the third case there existed an arteriovenous aneurysm between the internal carotid artery and the cavernous sinus." Green divides these murmurs into three classes as follows: Physiologic murmurs in the internal carotid artery, (1) dependent on partial stenosis of the carotid canal, (2) probably caused by a reduced vascular tension and dependent upon disturbances in the vasomotor system, and (3) pathologic murmurs from aneurysms of the vessels of the head.

Iglauer[14] presented a case in which an audible tinnitus was present for more than ten years. The sounds disappeared when pressure was applied over the carotid artery and also when the head was rotated to the side of the lesion. In the case to be presented here, objective and subjective sounds are present and disappear upon pressure and rotation of the head as described in the case reported by Iglauer.

In this case there is present an objective as well as subjective tinnitus, presumably due to a cerebral aneurysm on the right side. We wish to express our appreciation to Dr. Cyril B. Courville for his assistance in correlating the neurologic data.

REPORT OF A CASE.

Personal History.—The patient is a female, fifty-three years of age, married and a housewife. She had mumps and diphtheria as a child and after taking some serum for the latter developed an abscess of the abdominal wall. Some years ago she had a rather marked arthritis which disappeared after the extraction of teeth. There is quite a definite history of traumatic sphenopalatine neuritis which was undoubtedly caused by repeated intranasal surgery on the left ethmoid sinuses. Menstrual periods began at the age of eighteen and menopause was noted eight or ten years ago. The husband is living and well at the age of sixty. There are no children.

Present Complaint.—There is pain and tenderness beneath the left eye associated with irregular drainage from the nose. There is tinnitus and deafness in the right ear existent since April, 1934.

According to the patient's history and her belief, the trouble dated from 1926. At this time she was douching her nose with zonite and she noted that the solution

was apparently too strong, since the mucous membranes felt burned. The solution was immediately washed out, but since that time she has had definite trouble with her nose. Sinus trouble dates from this period, a gradual development of pain and tenderness in the region beneath the left eye and just adjacent to the nose being experienced. This pain has been relieved at various times by cocainization of the sphenopalatine ganglion.

On two occasions during the past year she underwent intranasal exploration of the left ethmoid areas. During this interval she noticed that her vision was definitely blurred, and this occurs whenever there is not free drainage from the nose. Coincident with this nasal difficulty there have been recurrent attacks of pain in the back, which the patient designates as lumbago.

Intracranial Symptoms.—The first symptoms which seem to be definitely associated with some pathology within the skull were noticed nine months ago. At this time the patient rather suddenly noticed a pounding noise in the right ear while at church. She did not know to what the noise was due, but she found that she was able to control it by pressure over the upper part of the right side of the neck. This abnormal sound has been more or less constant since and is described as a surging in the right ear. The sound varies considerably in nature and is described as a swishing or jetting sound, or at times when less marked it may be noticed as a low-pitched murmur on the inside of the head. Lately, when severe, this surging or swishing sound seems to radiate upward and extend over the vault into the left side. On these occasions the sensation is like swirling water. This sound is rhythmic in character and is synchronous with the heart beat. It is aggravated by any sudden movement or on throwing the head backward or turning it toward the left side. There has been a definite impairment of hearing in the right ear for some years, according to the patient, but the present difficulty seems to have aggravated the condition.

Status Presens.—The patient is a large, fairly well developed woman, past middle age, who does not present any unusual manifestations of ill health nor does she assume any position which is suggestive of any particular neurologic disease. Her illness has not incapacitated her in the carrying on of her household duties, but she is very much concerned about the pain in her nose and face and the unpleasant throbbing sensations in her head, which more recently are associated with a very marked throbbing sensation in the scalp at the top of her head. This rhythmic sound at times interferes with her sleep so that it is necessary to resort to sedatives.

Neurologic Examination.—There is no dilatation of the superficial vessels of the scalp. There is unusual tenderness of the scalp experienced to such a degree that the patient has found it necessary to discontinue the use of hairpins. The percussion note over the skull is not particularly high pitched, and examination of the cranial vault with a stethoscope discloses a characteristic murmur only intermittently. This murmur is audible to the examiner when a stethoscope is placed beneath the eye just adjacent to the nose on the left side. The cranial nerves were normal with the exception of the following:

Optic Nerve.—Patient has had a blurring of vision for the past two or three years which is indefinite in character. On one occasion recently, a band scotoma was apparently experienced by the patient, as she stated that on looking at an individual's face the lower part of the face and neck were not visible to her. Visual acuity was somewhat diminished and perimetric examination showed a moderate

concentric contraction of the fields. Examination of the fundi revealed the veins to be somewhat overfull, particularly in the left eye, with early tortuosity. No elevation of the discs was noted.

Trigeminal Nerve.—There is a definite history of pain in the left side of the face over the eye or at the emergence of the infra-orbital division of the left trigeminal nerve, associated at times with a creepy feeling over the left side of the face. This pain is referred into the left upper lip and toward the left ear, and also in the left parietal region of the skull and the left palate. There are no objective sensory or motor changes in the face or involving the muscles of mastication.

Acoustic Nerve.—The patient has had a definite tinnitus in the right ear for nine months which is concomitant with the heart beat. Tests for auditory acuity with whispered and conversational voice, with and without the Bárány apparatus and with the tuning forks, revealed an approximate decrease in acuity of twenty per cent in the left ear and seventy-five per cent in the right ear, which was chiefly of the conductive type, but definite damage in the higher registers was present. Tests of the vestibular apparatus showed normal reactions in all canals.

X-ray and Laboratory Findings.—Roentgenograms of the skull taken four months ago disclose a rather thin cranial vault with no evidence of increased intracranial pressure and no areas of erosion. The middle meningeal channels are rather large. There is one rather narrow Pacchionian depression at the junction of the two coronal sutures. The sella turcica is slightly larger than average, is round in type and the anterior clinoid processes seem to be rather sharp. The pineal body, which is faintly shown on the lateral view, on actual measurement seems to be displaced somewhat forward.

Examination of the blood showed: Hemoglobin, 100 per cent; red blood cells, 5,624,000; color index, 0.89; leucocytes, 7,900; polymorphonuclear neutrophiles, 54 per cent (nonfilament 3, filament forms 51); lymphocytes, 38.5 per cent; mononuclears, 3 per cent; eosinophiles, 4.5 per cent. The red blood cells were normal in size, regular in shape and took the stain evenly. No nucleated red blood cells were seen. Platelets were normal in number. Sedimentation time (Linzenmeier tube), 12 mm., 50 minutes, 18 mm., 80 minutes. The Wassermann test was negative. The blood pressure was taken on several occasions, the highest reading being 138-90.

There can be no question that the patient has a chronic suppurative disease in the accessory nasal sinuses on the left side, which is the contributing cause of the pain and distress which she experiences. The reference of the pain and clinical findings would lead one to believe that the sphenoid and post-ethmoid sinuses are involved on that side, producing some irritation of the sphenopalatine ganglion and giving rise to the characteristic symptom complex.

The head noise of which she complains, we believe is an entirely separate and distinct entity. In our opinion, this is due to an aneurysm of one of the large vessels at the base of the brain on the right side. The findings are somewhat vague insofar as exact localization is concerned, but the limitation of the murmur in the right

ear and the decreased auditory acuity on the right side point to its location as being in the right posterior fossa. In this case, the most probable site of origin is at the point of emergency of the posterior inferior cerebellar artery from the vertebral. An aneurysm in this region usually extends into the cerebellopontile angle on this side and gives rise to very definite auditory manifestations. This leads us to believe that an arteriovenous aneurysm of the cerebral substance can be definitely excluded.

There is very little to offer in the way of treatment of these intracranial lesions. We are of the opinion that ligation of the internal carotid artery would not prove of sufficient value to justify recourse to this procedure.

We have advised continued rest for a period of several months in the hope that the aneurysm may strengthen its walls sufficiently to prevent early rupture, inasmuch as the patient volunteers the information that any irritation or sudden movement accentuates the murmur.

Tinnitus aurium has been discussed by many otologists since it was first recognized, and of all the symptoms which the aurist is called upon to relieve, it is the most elusive and difficult to control. The most useful classification, based on the underlying lesions or disorders, is as follows:

1. Obstruction sounds, or noises due to occlusion or impaired mobility of some portion of the sound conducting apparatus.

2. Labyrinthine sounds, or noises due either to structural changes in the cochlea, or to alterations—either increase or diminution—of intralabyrinthine pressure.

3. Neurotic sounds, or noises due to abnormal irritability of the auditory nerve.

4. Cerebral sounds, or noises due to abnormal conditions acting upon the auditory centers in the cerebral cortex. (Auditory hallucinations.)

5. Blood sounds, or noises produced by the blood current in vessels in or near the ear, and due to either disturbances in the local or general circulation or to abnormalities in the size, shape or position of the vessels.

The first four types cause subjective tinnitus and the last one a tinnitus of both objective and subjective types.

An objective tinnitus—that is, one that can be heard by the examiner through the auscultation tube—may be caused by two conditions. The first is a clicking or snapping sound caused by a rhythmic, spasmodic contraction of the muscles related to the eustachian tube, which are the levator palati, tensor palati and tensor tympani. These sounds are not synchronous with the beat of the heart, however, and so are not of vascular origin, as the lesion in the present case undoubtedly is. The vast majority of cases of objective tinnitus are of vascular origin, and there are many pathologic possibilities, of which the following is a fairly complete list. Valvular lesions in the heart itself, abnormalities of the carotid artery, the jugular bulb or of the blood vessels near the affected ear may be the cause. In this case there is no valvular lesion of the heart. Abnormalities of the carotid artery occur rarely, but when present are centrally located near the pituitary body. If the objective tinnitus were caused by an abnormality of the jugular bulb or internal carotid artery, such as an unusually close proximity to the internal ear due to a dehiscence, the tinnitus would always have been present. In this patient the tinnitus is of but a few months' duration. Traumatic aneurysms of the temporal or occipital arteries may cause objective tinnitus which disappears on ligation of the affected vessel. There is no history of trauma in this case.

As is well known, the lateral sinuses on the two sides frequently vary in size, and it might be assumed that a large lateral sinus discharging into a small jugular bulb could set up an audible bruit, but in this event the resulting tinnitus would have been present during at least the greater part of her life. A brain tumor of vascular type must of course be considered, but as yet there are no definite signs to indicate this type of lesion.

The fact that rotation of the head will sometimes cause a cessation of the tinnitus and that sometimes it is necessary to turn the head from side to side to initiate it is an interesting feature of this case. It would seem that only slight changes in vascular pressure alter the mechanics of the situation.

This lesion is in all probability of unilateral occurrence because the tinnitus is present only in one ear. The fact that practically all other causes of objective and subjective tinnitus can be eliminated points to the probability that the cause is a cerebral aneurysm. The point of greatest possibility in these aneurysms is the juncture of the vertebral with the right cerebellar arteries.

BIBLIOGRAPHY.

1. Sands, Irving J.: Aneurysm of the Cerebral Vessels. Arch. of Neur. and Psychiatry, 21:1, 1929.

2. Schmidt, Max: Intracranial Aneurysms. Brain, A J. of Neur. MacMillan & Co., Lmt., 13:489-492, 1930.

3. Green, J. Orne: Objective and Subjective Systolic Murmurs in the Ears: A Contribution to the Study of Brain Murmurs. Tr. Amer. Otolog. Soc., 2:267, 1875-1881.

4. Von Hofman, E.: Aneurysm of Basilar Artery and Its Rupture as Cause of Sudden Death. Wien. Klin. W., 7:823, 848, 867, 886, 1894.

5. Lebert, H.: Berl. Klin. Woch., III, 1866.

6. Busse, Otto: Virchow's Arch. 299:178, 1921.

7. Wichern, Heinrich: Muenchen. med. Wchnschr., 1911. Deutsch. Ztschr. f. Nervenh., 44:220, 1912.

8. Beadles, C. F.: Aneurysm of the Larger Cerebral Vessels. Brain, 30:285, 1907.

9. Weil, Arthur I.: A Case of Objective Tinnitus Auri. Laryngoscope, 14, 1904.

10. Kafka, M. Martyn: Tinnitus Aurium: Etiology, Differential Diagnosis, Treatment and Review of Twenty-five cases. Laryngoscope, 44:515, 1934.

11. Poorten: Monatschr. f. Ohrenh., 1878.

12. Moos: Arch. f. Augenh., IV, 174.

13. Grüber: Lehrbuch d. Ohrenh., p. 628.

14. Iglauer, Samuel: Objective Tinnitus Aurium with Report of Four Cases. Arch. of Otolaryng., 18 (July), 1933.

XLIV.

IRRADIATION THERAPY OF MALIGNANT TUMORS OF THE ORAL CAVITY, EYE, EAR, NOSE AND THROAT.*

CHARLES L. MARTIN, M. D.,

DALLAS.

Cancer therapists have for years dreamed of a remedy possessed of the power to relentlessly track down and destroy every cancer cell in the body without damaging the normal tissues. When radiant energy was first shown to exert a more harmful effect on certain neoplastic cells than on the adjoining normal structures our hopes were aroused and the desired medium seemed to be at hand. However, clinical experience soon revealed a lack of regression in many of the malignant tumors treated with the earlier technics. These new powerful rays did not seem to have a selectivity great enough to save the normal structures when doses capable of killing the less malignant types of cancer cells were given. It became evident that unless this selectivity could be improved the uses of the new method would be much restricted. Progress has been slow and technics will no doubt be definitely improved in the future, but the field of irradiation therapy has been broadened by the evolution of the following principles:

(1) A total of three to twelve minimum erythema doses of irradiation is needed for the complete extermination of the various grades of epidermoid carcinoma.

(2) Recovery is more rapid and the surrounding tissues show a quicker return to normal when the treatment is administered over a prolonged period (usually seven to twenty-one days, at present).

(3) An attempt should always be made to produce a cure with the first series of treatments, since the second attempt is never so effective.

(4) Short wave lengths produced with heavy filtration lessen the injury to surrounding normal tissues, but this protection is not essential in the treatment of superficial lesions.

---

*Professor of Radiology, Baylor University Hospital.

Presented before the meeting of the Mid-Western Section of the American Laryngological, Rhinological and Otological Society at Dallas, Texas, January 29, 1935.

(5) Infections and other sources of irritation definitely retard healing after irradiation.

TECHNICS.

The earliest therapy was done with x-rays having a relatively long wave length and low penetrating power, and naturally the good results were limited to malignant tumors of the skin. Pusey,[1] who had no accurate measuring devices available, published case reports with illustrations showing patients cured of squamous cell cancer of the lip more than twenty-five years ago. Although his technic utilized many of the principles elaborated above, it has never been popular with dermatologists. However, it is still a very valuable procedure, and for superficial tumors measuring an inch or less in diameter it may still be considered the method of choice. It will hereafter be designated as the "superficial x-ray technic." The factors used by us are 87 kv. peak, 5 ma., a target skin distance of ten inches, and a filter of 0.5 mm. of aluminum which with our apparatus produce a mild erythema over an area one inch square with an exposure time of five minutes. The size of the area treated is of course quite important, since this dose produces 555 r with back-scattering over a circular area ¾ inch in diameter, 640 r over an area 1½ inches in diameter, and 666 r over an area 2 inches in diameter. In practice one or two erythema doses is given at each visit and the treatments are repeated every second day until a series of six or more is administered.

The shorter wave lengths for deep therapy were first generated at 130 to 140 kv. peak with filters of 3 or 4 mm. of aluminum. Just after the World War, German physicists developed machines generating 200 kv. peak and advocated filters of ½ to 1 mm. of copper. Elaborate charts were produced showing exactly how much radiant energy could be delivered to various points within the body and complicated plans for cross-firing were worked out. All of these changes tended to improve the therapy of deep seated tumors, but unfortunately the theory was advanced that the delivery of a single erythema dose to all parts of a malignant tumor by the new method would cause its complete regression. Although no clinical evidence was ever produced to support this theory, the single "massive dose" plan has been carried out in many American clinics ever since its introduction from abroad.

In 1925, Pfahler proposed his saturation method, which constituted an important step forward in the evolution of efficient depth dosage. He adopted the theory of Kingery, which states that a dose

of x-rays decays in accordance with the values indicated by a logarithmic curve. An erythema dose was rapidly built up in a few days and small doses were then administered every few days thereafter. These smaller doses were calculated from the curves and were supposed to be just large enough to return the decaying dose to its original level. This plan really succeeded in delivering about 2½ erythema doses to a single area in one month and was therefore a definite improvement on the massive dose method.

There has been a tendency in this country to build larger machines with higher voltages in an effort to obtain shorter wave lengths and thereby improve the selective action of the rays. Such a machine operating at 700 kv. peak has been in use at the Memorial Hospital in New York since 1931, and a few others have been installed elsewhere, but although the results obtained indicate some increased efficiency they have not as yet seemed to justify the huge expense involved.

The most important advance in deep therapy has come from France, where it has been shown, both experimentally and clinically, that very large doses of x-rays may be passed through normal tissues without irreparable damage, provided they are properly fractionated and prolonged over a sufficient period of time. Experiments performed by Regaud and Ferraux[2] with rams' testicles show that although the fractionization spares the adult cells of the normal tissue, the effect on the more embryonic cells is almost the same as it would have been if the dose had all been given at once. The increased selectivity produced by this method has been utilized clinically by Coutard[3] for a period of about fourteen years, and his results testify to its efficiency. Although Coutard believes that a low intensity (6 r per minute) is essential to the success of his method, good results have been obtained in this country with much higher intensities by Martin and McNattin[4] and the author.[5]

Radium technic has advanced in a somewhat similar fashion. Interstitial work was first done with relatively large amounts of the salt in needles having thin walls of steel which allowed many of the more irritating rays to pass through. Regaud[6] began to advocate heavy filtration, low intensity and long periods of radiation some fifteen years ago. In 1926, Failla[7] described the gold radon seed which carries out Regaud's principles except that the filtration is not maximum. These implants are advantageous because of their small size and because they may be left permanently in place. Unfortunately their preparation requires a very expensive set of equipment. In 1927 Evans and Cade[8] advocated radium element needles that are

both economical and efficient. They may be used as a substitute for radon seeds which radiologists in Texas obtain only from New York City. These needles are made of platinum-irridium and their dimensions are given in Fig. 1. Details relative to their use have been published in previous articles.[9] [10] It has been shown that a cubic centimeter of squamous cell carcinoma will regress if 0.6 mg. of radium filtered with 0.5 mm. of platinum be placed at its center for a period of seven to eight days, and for this reason all of the needles contain 0.6 mg. of radium per centimeter of active length.

It is obviously impossible to discuss the detailed use of the above technics in the treatment of all the many types of malignant tumors occurring about the head and neck in a paper of this sort, and a few of the regions yielding the most promising results will therefore be singled out.

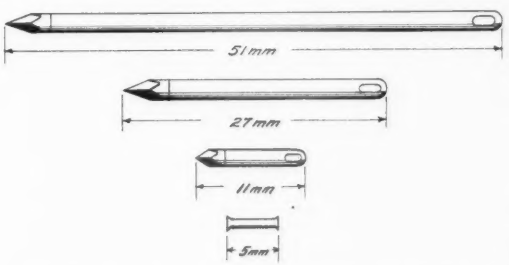


Fig. 1. Relative sizes of weak platinum radium needles and a radon seed. The needles contain 0.6 mg. of radium per cm. of active length and have wall thicknesses of 0.5 to 0.6 mm. The seeds are made of gold or platinum and have walls 0.3 mm. thick.

CANCER OF THE LIP.

Cancer of the lip has in our experience proven a very favorable lesion for radiation therapy. When the tumor measures no more than one inch in diameter it is treated by the superficial x-ray technic because of the superior cosmetic results that may be obtained. The treatments are administered to the growth and a narrow margin of adjoining tissue on alternate days for a period of about two weeks, and a total of six to twelve erythema doses is given, the variation in dosage depending upon the thickness of the tumor. A severe reaction follows this procedure, but healing is usually complete in six to eight weeks, and the lack of scarring is evident in the cases illustrated in Fig. II. When the tumors are large and extend into the cheek at the corner of the mouth, or when they are deeply indurated, the weak radium needle technic described under the heading of intra-

Fig. 2. Squamous cell carcinomas of the lip treated with the superficial x-ray technic which have remained well for periods varying from four to six years without neck surgery. Note the absence of scarring after healing occurs.

oral cancer is used. The care of the neck containing adenopathy will be discussed later on, but our experience has led us to the conclusion that a routine block dissection is not necessary when no glands are palpable. In 1931, J. M. Martin[11] reported a series of 108 cases treated by the above method between 1906 and 1925. None of these patients had palpable glands at the time of treatment, and 104 of them had remained well for periods of five years or more. Surgical results covering routine block dissections are certainly no better, and it seems difficult to avoid the conclusion that most operations in cases of this sort are unnecessary.

INTRAORAL CANCER.

Tumors involving the cheeks, alveolar margins, anterior tongue and floor of the mouth are suitable for interstitial radium therapy because of their accessibility. Radon seeds are quite efficient in these regions, but the heavily filtered weak radium element needles are also very useful. It is desirable to place these needles beneath the tumor bed and beyond the growing edges, spacing them about one cm. apart. Each needle is sutured in place and a guy thread is brought outside the mouth and attached to the cheek so that it can not be lost or swallowed. Most implantations are done under local anesthesia. After the work is completed radiographs are made in order that the needle pattern may be studied. When the radium distribution is not correct the needles may be easily rearranged. In most cases the treatment is continued for seven days, during which time it is necessary to pay strict attention to oral hygiene. The mouth is irrigated frequently with warm bland solutions and the involved area is painted daily with 5 per cent mercurochrome. Use of a spray containing liquid albolene at regular intervals is desirable. Pain is usually not a troublesome symptom after the first twenty-four hours. Following removal of the needles the tumor usually melts down rapidly and a white membranous reaction appears. This change is a superficial one and is in no way similar to the painful sloughs formerly produced by steel needles. The membrane clears away after a few weeks, leaving a clean granulating surface, and healing occurs in about two months. A soft pliable scar which interferes very little with function results. In tongue lesions taste is not interfered with and mobility is unimpaired. Bony structures situated near the sources of radiant energy show little or no damage, although occasionally flakes of the cortex may separate. Large tumors involving the hard palate, such as the one shown in Fig. 4, have disappeared promptly without any apparent bone damage. A review of our rather limited material was published in a recent article.[12] All of

Fig. 3. Large squamous cell carcinoma Grade II which grew in the left cheek and pushed the molar teeth out of their sockets. The radium needles shown in the radiograph remained in place seven days and were augmented by 1500 mg. hours delivered externally with 50 mg. radium packs and a single erythema dose of deep x-rays delivered to the side of the neck. There is no palpable or visible evidence of carcinoma made out at the end of two years and three months, at which time the lower photograph was made.

the primary lesions, except a few in which local irritation played a big rôle, showed satisfactory regression. Forty per cent have remained well for one or more years. Since our experience with weak radium needles is of only about four years' duration we can provide no five-year statistics as yet.

CANCER OF THE EYE.

Malignant tumors frequently appear on the eyelids and at the inner canthus, where it is very important to preserve function. Fortunately most such lesions are very radiosensitive, and the main problem is the protection of the normal structures of the eye. Both the conjunctiva and the lens can be seriously damaged by large therapeutic doses. The superficial x-ray technic produces rays of minimum penetrating power, and it is therefore best suited for this work. At each visit an oval lead shield is slipped under the cocainized lids, after which the lesion is isolated with other lead shields and intensively radiated. When healing occurs all of the normal tissue is conserved and usually no cosmetic surgery is necessary. Tumors near the nose frequently extend inside the orbit, where masses of considerable size can be palpated just inside the orbital rim. These extensions can be satisfactorily treated by the insertion of the 1.33 mg. radium needles at 1 cm. intervals for a period of seven days. No eye damage results if the needles are planted near the wall of the orbit.

The superficial x-ray technic is particularly well suited to the treatment of malignant tumors of the ocular conjunctiva. Grier[13] reported eleven such cases successfully treated with x-rays ten years ago. At each visit it is our custom to fit a carefully prepared lead shield over the cocainized eyeball with lid retractors in place. A hole is cut in the shield to fit the lesion and an erythema dose is administered every second day until six such doses are given. The reaction is slight and no eye damage results. Photographs of a large carcinoma of this type before and after treatment were published in a recent article.[14] This patient has been well for more than two years and his vision is normal on the treated side.

It is our feeling that radiation has no place in the treatment of primary intra-ocular tumors. However, the Coutard plan of administering deep therapy is of definite value as a postoperative procedure, particularly when metastases seem likely.

CANCER OF THE EAR.

Small superficial carcinoma of the skin of the ear respond well to minimum total amounts of superficial x-ray therapy. However, the use of large doses in this region is hazardous because a very stub-

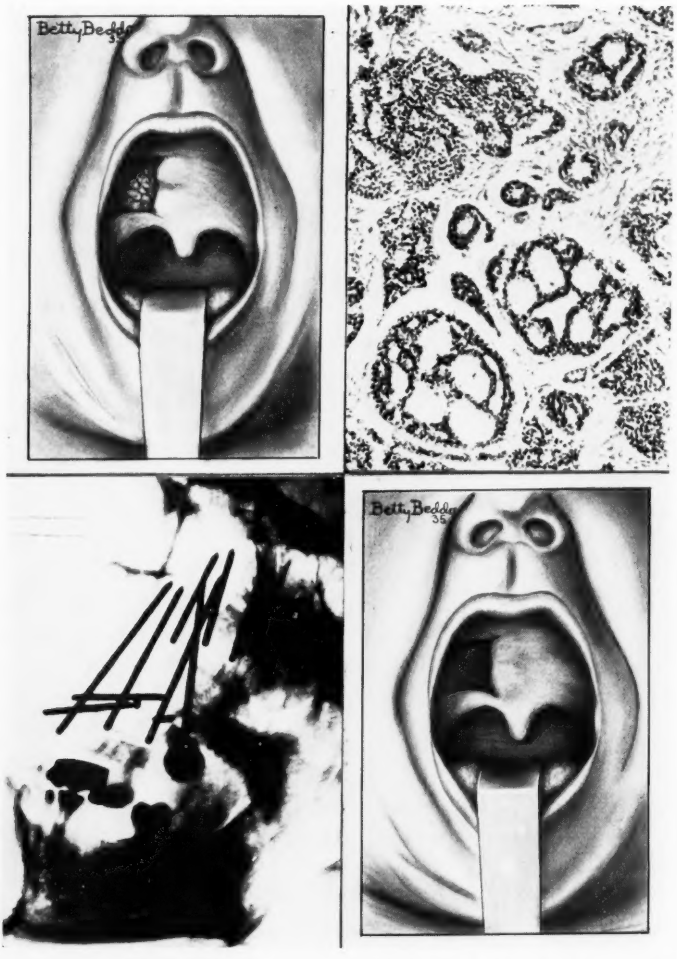


Fig. 4. Recurrent adenocarcinoma Grade I of hard palate which recurred both in the palate and in the antrum following operation as shown in the upper drawing. The lower sketch shows the absence of all visible evidence of the tumor four months after the radium needle implantation shown in the radiograph. The needles remained in place for seven days.

born and painful reaction is frequently set up. This peculiarity has been attributed to the effects of radiant energy on cartilage. It seems best, therefore, to treat well advanced lesions by complete removal of all or part of the ear. Contrary to the belief of many surgeons, this procedure is frequently not curative, since deep invasion often extends down into the tissues about the parotid and below the auricle. It is our custom to remove the auricle, after which weak radium needles are placed around the ear and in all suspicious places for a period of a week. A good result obtained in this way is shown in Fig. 5. When massive tumors involve the ear and the side of the face, doses ranging between 4000 and 5000 r administered during a period of approximately three weeks, by the Coutard plan, often produce striking improvement. The number of postoperative cases of cancer of the ear with deep metastases that have appeared in our clinic make it evident that more attention should be given this subject.

CANCER OF THE PHARYNX AND LARYNX.

With the advent of interstitial radium therapy various plans were worked out for implanting the relatively inaccessible tumors of the pharynx and larynx, and radon seeds were obviously better than needles for this type of work. Some excellent results were obtained, but much difficulty was experienced in evenly irradiating the larger tumors and untoward reactions in the cartilage of the larynx limited the dosage in this region. The internal irradiation was augmented by radium packs and a massive dose x-ray technic, but the external irradiation did not assume a rôle of major importance until Coutard elaborated his method whereby large, efficient doses could be delivered to the midline of the throat without the production of serious injuries to the normal tissues. This made it possible to rely on deep x-rays alone, and the present tendency is to eliminate implanted radium in the treatment of the deeply seated tumors.

Fortunately the carcinomas of the pharynx are much more radiosensitive than their histology would indicate, and they do not require doses as large as those needed for the epithelial neoplasms of the mouth. Coutard felt that he obtained his best results with 190 kv. peak, 2 mm. of zinc filter, 5 ma. of current and a target skin distance of 50 cm. Areas measuring not more than 15 cm. on a side were laid out on the lateral aspects of the neck overlying the involved region and treated on alternate days. Each dose ranged from 200 to 300 r measured in the beam without back scattering, and the treatment was continued for three or four weeks. The total dosage over each skin area amounted to 3000 r to 5000 r, and about 3500 r was

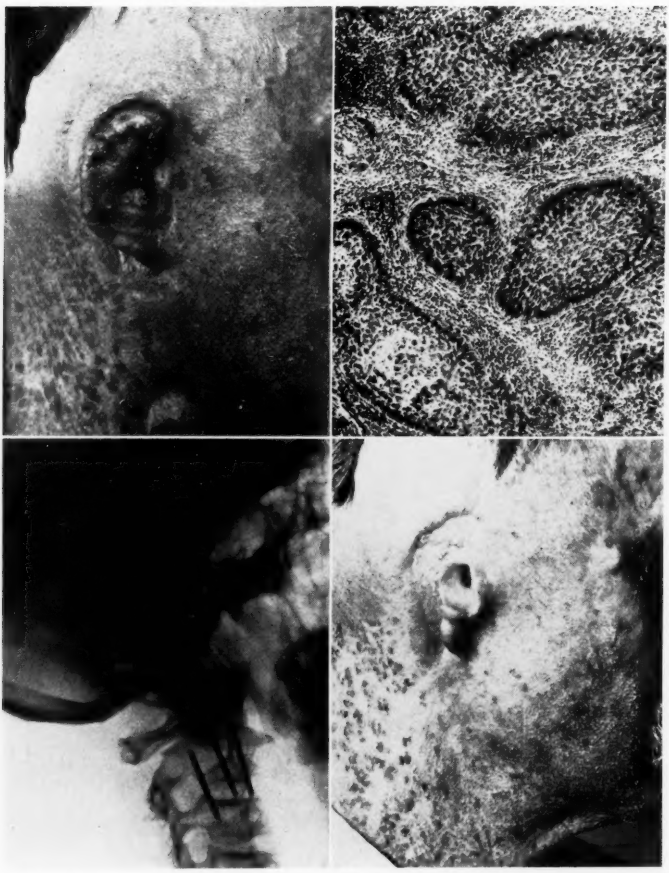


Fig. 5. Squamous cell carcinoma Grade III of the upper auricle with deep involvement of the surrounding tissues treated with radio knife resection and the implantation of weak radium needles for seven days. There was a palpable gland in the neck. The lower photograph made one year and two months later shows perfect healing. No palpable evidence of malignancy could be made out.

delivered to the tumor. As the treatment progressed the throat became very sore and finally a superficial white membrane appeared on the surface. As the mucous membrane reaction subsided the skin surfaces became red and finally much of the epithelium disappeared in the treated areas, leaving a moist surface on which small patches of epithelium often remained. This reaction also healed readily, so that there were few sequelæ present after two months had elapsed. The malignant tumors melted away as the reaction in the throat appeared, and many of them were permanently controlled. Between 1920 and 1926 Coutard treated 212 malignant tumors of the pharynx and larynx, many of which were inoperable, and 20 per cent of this group remained symptom-free for a period of five years.

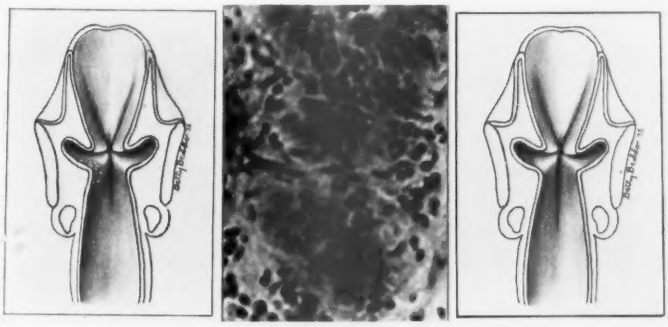


Fig. 6. Early epidermoid carcinoma of the vocal cord treated with deep x-rays by delivering 4250 r. to the sides of the neck during a period of 21 days. Two months later after the reaction had disappeared the laryngologist reported only a slight scarring which the artist failed to indicate in the sketch on the right.

About five years ago Lenz, Coakley and Stout[15] began the treatment of a series of thirty-one cases of cancer of the pharynx and larynx, using 200 kv. peak, 8 ma., 1.86 mm. of copper and 1 mm. of aluminum filter and a target skin distance of 50 cm. Fields were laid out, usually on the sides of the neck, and daily doses of 400 r to a single area were given until each of them received from 2800 to 4000 r. Approximately 45 per cent of these cases remained clinically well for periods varying from nine months to twenty-four moths of observation.

In 1931 Martin and McNattin adopted a technic utilizing 200 kv. peak, a filter of 0.5 cm. copper and 2.5 al., 4 to 30 ma. of current and a target skin distance of 50 to 60 cm. Malignant tumors of the tonsil, soft palate, pharyngeal walls, nasopharynx, extrinsic and

intrinsic larynx, base of the tongue and floor of the mouth were treated. Circular parts, 7 cm. in diameter, were used for most of these patients. Out of a series of 140 cases, 29 per cent have remained free of disease for periods varying from 1¾ to 2½ years. In a few cases the treatment was augmented by radon seeds implanted in metastatic cervical glands.

For 1½ years we have used a technic similar to that of Martin and McNattin. Our enthusiasm was aroused when we observed the rapid disappearance of a very large prickle cell carcinoma of the face after it had received 3600 r in twelve equal doses given over a period of fourteen days. An effort has been made to deliver somewhat similar doses to tumors in the throat, and the immediate results have been most encouraging. This method can also be used to great advantage in treating advanced carcinoma of the sinuses and nasal passages where the disease is too inaccessible for radium therapy. After observing the cases treated by Merritt in Washington, D. C., with 220 kv. peak, a filter of 2 mm. copper and 1 mm. aluminum, 20 ma., and a target skin distance of 50 cm., we have installed a similar set of apparatus and have adopted his technic. Of the many modifications of the original Coutard plan, this one impresses us as the most efficient at the present time.

The reader is no doubt thoroughly impressed with the fact that deep therapy is now in a state of progressive improvement. The method is a dangerous one, which should be handled only by an experienced radiologist, but if he be very careful and not too conservative he will obtain some surprisingly good results and very few troublesome sequelæ.

CERVICAL METASTASES.

The stumbling block in the path of all those who treat cancer about the head is the care of metastatic cervical lymph nodes. Neither the surgeons nor the radiologists can point with any great pride to the results obtained in this field. For practical purposes all cases may be divided into three classes, namely, those having no palpable lymph nodes; those having palpable nodes that are considered operable, and those having inoperable metastases in the neck.

The first group usually occasions the greatest controversy because many of our surgical confreres feel that a block dissection should always be done. In cancer of the lip we are opposed to such surgery as a routine procedure, because we are convinced by our own statistics as well as those of Duffy[16] and by the small number of

malignant glands found by pathologists in the resected tissue, that the operation is unnecessary in most of the cases on which it is done. At the time of treatment the importance of the appearance of lymph nodes in the neck must be impressed on the patient, and he should be examined at regular intervals for a year or more. In this way the few who develop metastases may be submitted to operation at a favorable stage. The intraoral tumors metastasize much earlier than do those on the lip, and block dissection may be advised at the time that the primary lesion is treated. However, the patient frequently does better if the operative procedure is delayed several months, and a fair number of our cases have remained well without neck surgery. It is our custom to use heavy x-ray therapy administered in fractional doses over the neck as well as implanted long weak radium needles, but their value is not easily estimated.

When operable cervical glands are present at the time that the primary lesion is treated the operation should be done. Unfortunately all surgeons do not establish operability in the same manner, and radiologists see many patients with intractable malignant infiltration in the neck following ill advised surgery. Quick's[17] criteria are based on an ample experience and form a reliable guide. He states:

> "Unilateral neck dissections under local anesthesia are done in cases of adult type epidermoid carcinoma presenting clinically involved unilateral nodes with intact capsules and in the presence of a primary growth either controlled already or favorable from that standpoint. Bilateral involvment of nodes is considered an indication of inoperability. Perforation of the node capsule is an indication of inoperability. * * * We regard the metastatic nodes of transitional cell carcinomas, in fact all cellular epidermoids, as being more amenable to radiation therapy than dissection."

In inoperable cases having nodes of moderate dimensions Quick inserts radon seeds into the individual nodes after exposing them at operation. The small platinum radium needles may be used in a similar manner. When the masses in the neck reach a considerable size satisfactory regression can frequently be obtained by inserting parallel rows of long platinum radium needles in the base of each tumor at 1 cm. intervals for a period of seven or eight days. This procedure can be safely augmented by doses of deep x-rays totaling 1800 to 2000 r and given at the rate of 300 r per day. The more embryonic tumors of the type shown in Fig. 7 show an excellent response to this form of therapy, and even those containing more adult cells are reduced in size and may remain quiescent for many months. These patients frequently show a rapid improvement and may obtain complete relief of pain for long periods.

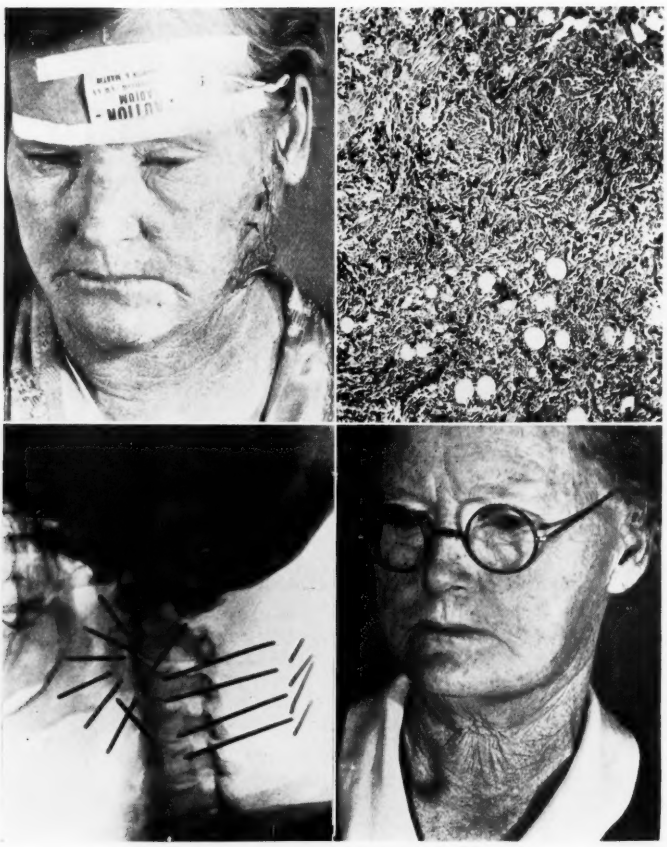


Fig. 7. Diffuse undifferentiated infiltrating carcinoma forming a mass three inches in diameter below the ear and extending around the back of the neck to the midline. No primary could be found. Weak radium needles were implanted for seven days as shown in the radiograph. The lower photograph was made a year later when no palpable evidence of carcinoma could be made out.

SUMMARY.

1. Many primary epidermoid carcinomas of the lip, mouth, pharynx, larynx, eye and ear can now be completely eradicated with radiation alone.

2. Although rather severe temporary reactions frequently are produced at the time of treatment, the excellent cosmetic results finally obtained make them justifiable.

3. Cervical metastases are best handled by a judicious selection of the surgical or radiation technic best suited to the individual case.

BIBLIOGRAPHY.

1. Pusey, William Allen: The Principles and Practise of Dermatology. D. Appleton & Co., New York, 1907.

2. Regaud, Cl., and Ferraux, R.: On the Diverse Reactions of the Tissues Treated by Roentgen Rays in Relation to the Time Factor, and on the Relativity of the Biologic Dosimetry in Roentgen Therapy of Malignant Tumors. Ztsch. f. krebs forsch 32:10-26, 1930.

3. Coutard, H.: Roentgen Therapy of Epitheliomas of the Tonsillar Region, Hypopharynx and Larynx from 1920 to 1926. Am. J. Roentgenol. 27:313-331, (Sept.) 1932.

4. Martin, H. E., and McNattin, R. F.: The Treatment of Cancer of the Pharynx, Tonsil and Extrinsic Larynx by Divided Doses of External Radiation. Am. J. Roentgenol. 32:717-729, 1934.

5. Martin, C. L.: Modified Coutard Technic in Deep X-ray Therapy. Texas State J. of Med. 30:207-210, 1934.

6. Regaud, Cl.: Radiotherapy of Cancer of the Tongue. Paris Med. 11:265-271, 1921.

7. Failla, G.: The Development of Filtered Radon Implants. Am. J. Roentgenol. 16:507-525, 1926.

8. Evans, A., and Cade, S.: Cancer of the Tongue. Brit. J. Surg. 15:55-70, 1927.

9. Martin, C. L.: Small Radium Needles Versus Radon Implants. Am. J. Roentgenol. 27:240-247, 1932.

10. Martin, C. L.: Treatment of Malignant Tumors. Advantages of Weak, Heavily Filtered Radium Needles. J. A. M. A. 99:1587-1591, 1932.

11. Martin, J. M.: Radiation Therapy in the Treatment of Cancer of the Mouth and Lips. Radiology. 16:881, 1931.

12. Martin, C. L.: Carcinoma of the Lip and Mouth. Radiology. 22:136-146, 1934.

13. Grier, G. W.: The Treatment of Malignancy of the Eyeball. Radiology. 4:125-127, 1925.

14. Martin, C. L.: Squamous Cell Carcinoma of the Skin. Am. J. Roentgenol. 28:728-736, 1932.

15. Lenz, M., Coakley, G. C., and Stout, A. P.: Roentgen Therapy of Epitheliomas of the Pharynx and Larynx. Am. J. Roentgenol. 32:500-507, 1934.

16. Duffy, J. J.: Cervical Lymph Nodes in Intraoral Cancer. Radiology. 9:373-379, 1927.

17. Quick, D.: Interstitial Radiation in Metastatic Cervical Nodes of Epidermoid Carcinoma. Annals of Surgery. 93:380-390, 1931.

XLV.

CONFUSING SYMPTOMS IN ACOUSTIC NERVE TUMORS.*

CLAUDE T. UREN, M. D., AND

B. CARL RUSSUM, M. D.,

OMAHA.

When a tumor of the nervus acusticus presents the classical syndrome of primary auditory symptoms followed by signs pointing to involvement of the adjacent cranial nerves, occipitofrontal pain and stiff neck, ataxia and signs of compression of the medulla, diagnosis is easy. But a large tumor in the angle may produce distortion of the brain stem and resulting confusing symptoms. It is to this group of confusing symptoms that we wish to draw attention.

So-called "medullary symptoms" are seen in an appreciable number of patients with cerebellopontile angle tumors. These symptoms are due to involvement of the motor and sensory pathways by pressure and distortion.

Numbness and twitching of the arm or leg on the homolateral side of the body were observed in seven of the twenty-nine verified cases reported by Cushing.[1] Symptoms of involvement of sensory and motor tracts leading to the side of the body contralateral to the tumor are also met with and are more easily understood as due to direct involvement of the pyramidal tract before decussation. That an angle tumor may be of sufficient size to involve the pyramidal tract of the opposite side before it decussates and thereby lead to homolateral symptoms is not so generally appreciated, and recognition of the possibility will prevent mistakes in the interpretation of the tumor syndrome.

If one sees the patient after the general picture is obscured by the dominance of some one symptom, such as contralateral hemiparesis of the body, a misinterpretation may follow and a diagnosis of cerebral tumor may be made unless careful attention is paid to the chronology of the symptoms.

Involvement of adjacent cranial nerves due to pressure and distortion is common, but irritation or palsy of contralateral nerves may

*From the Departments of Otology and Pathology, the Creighton University School of Medicine and St. Joseph's Hospital, Omaha, Neb.

give misleading symptoms and even lead to erroneous tumor localization. The following case exemplifies the importance of the chronology of the symptoms in the presence of confusing medullary symptoms and the involvement of a contralateral cranial nerve.

REPORT OF CASES.

CASE 1.—R. 297. Left acoustic tumor with neighborhood symptoms, marked intracranial pressure, confusing medullary symptoms and contralateral vestibular nerve involvement. Decompression and ventriculogram. Death. Autopsy.

Mrs. L. C., a white housewife of 24, admitted on the service of Dr. G. W. Dishong because of blindness and weakness of the left side. Her past history is unimportant.

About eighteen months ago she gradually grew deaf in the left ear.

About five months ago she noticed her left leg was weak. Then a numbness and occasionally cutting pains developed in the leg. With the onset of the pain she noticed that her vision was failing and now she can only distinguish light from darkness. Of late there has been weakness in both legs and walking is difficult because of weakness. There has been no headache or vomiting and she is mentally clear.

She is a small, frail woman, thirty pounds underweight. There is left sided deafness and she is almost blind in both eyes.

Positive Neurologic Findings: (a) General Pressure. Bilateral choked disc, with four diopters swelling on the right and five on the left. Poor vision both eyes. In the left eye there is a small white area at the temporal border of the disc, apparently the result of a former hemorrhage. The branches of the central retinal veins are engorged and the arterioles on the disc are very tortuous in each eye.

(b) Localizing. (1) Cerebellar. Gait is uncertain, apparently from weakness. Falls to the left on attempting to stand. Slight ataxia of all extremities, most marked on left. Spontaneous nystagmus to the right of second degree. Nystagmus present on extreme rotation of eyes in all cardinal directions. (2) Extracerebellar. Cerebral nerves. Eighth, cochlear branch. Complete deafness for conversation, left ear. Weber lateralized to the right ear. Rinne test positive on left. Schwabach test minus 10 for left ear, normal for the right. C No. 1 fork minus 30 left ear, normal on right; C No. 4 fork not heard left ear, normal on right.

Vestibular branch. Bárány's test: Douching of each ear with water at 68° for five minutes produced no nystagmus and no past pointing.

Twelfth. Tongue deviates to right on protrusion. (3) Medullary. Weakness and atrophy of left lower extremity. Numbness and pain in left leg. Weakness in right leg.

Right patellar reflex more prominent than left.

Presumptive Diagnosis: Intracranial lesion, probably of the left cerebellopontile angle and of such size as to cause pressure on the vestibular branches of the eighth nerve of the opposite side.

Result: Death followed decompression and ventriculogram.

Postmortem Examination: "In the left cerebellopontile angle there is a spherical blue-gray tumor mass approximately 3 cm. in diameter. It lies in a large indentation on the left lobe of the cerebellum and the left side of the pons. The rest of

the pons and the medulla are pushed to the right. The left lobe of the cerebellum is also pushed downward. The left eighth nerve, identified at its origin, is stretched downward and leads into the tumor capsule, where it is lost. There is distortion of the left ninth, tenth and eleventh cranial nerves.

"On section the tumor mass is quite vascular and is gray streaked and mottled with red areas."

Histological Examination: The basic structure of this tumor is fibrous. In some areas the nuclei are quite numerous, while in others they are very scanty and the intercellular substance is fused into hyalin material. Contrary to the usual finding in these tumors, this one is very vascular, containing numerous large veins filled with clotted blood, and in several regions there is thrombosis of the veins. In some areas there is evidence of old hemorrhage, with many phagocytes loaded with blood pigment. In many of the small arteries there is rather extreme hyalin

Fig. 1. Left pontile angle tumor from Case 1.

change, and this hyalin change involves the surrounding tissue for a distance, in many instances as great as the diameter of the blood vessel. There are small lymphoid collections and larger mononuclear cells about the vessels and scattered more diffusely through the tissue spaces, especially near the surface of the tumor, where the evidence of old hemorrhage is more pronounced. There are a few areas of reticular edematous tissue adjacent to some fibrous bundles.

Diagnosis: Acoustic neurinoma.

In Cushing's[1] thirty cases there was contralateral cerebral nerve involvement in fourteen. None of the explanations advanced for such an occurrence is satisfactory. Displacement of the pons and the medulla, pressure of the nerves against the sharp edge of the dura at their emergence, or pressure of the pons and the medulla against the base of the skull on the opposite side, are the usual explanations, but Cushing[1] is not willing to accept any of them as applying in all cases.

In the one case above cited the vestibular portion of the eighth nerve on both sides was paralyzed, whereas the tumor was on the left side.

This is a distinctly unusual finding and there is no mention of contralateral vestibular involvement in Cushing's tabulations of his thirty cases.

The ear examination in this case is of interest because of the results obtained from tests of both the cochlear and vestibular branches.

While the patient was deaf to conversation in the left ear, yet there remained some hearing for tuning forks of low vibrations.

There was no response from stimulation of the vestibular nerve of either ear. Complete loss of function of the vestibular nerve on the affected side and only partial loss of function of the cochlear nerve may be interpreted as corroborating Henschen's[2] postmortem observation that acoustic tumors originate on the vestibular portion of the eighth nerve.

According to Jones and Fisher,[3] complete loss of function of the vestibular nerve on the homolateral side, together with a loss of function of the vestibular fibers from the vertical canal of the contralateral labyrinth, is most often found in pontile angle tumors and they have designated these findings as an angle syndrome.

According to Cushing[1] and his associates, the response to vestibular stimulation in cases of eighth nerve tumors is very characteristic.

Irrigation of the ear on the affected side leads to very little, if any nystagmus, to very slight reaction movements, to no vertigo or nausea. Irrigation of the opposite ear results in definite fine or coarse nystagmus, in well defined reaction movements and in very slight or no dizziness.

Cushing[1] further states that on testing hearing the patient as a rule is found to be deaf on the homolateral side. However, he mentions that complete loss of vestibular reactions with some retention of hearing may also be seen but is less common.

The caloric test of Bárány for the evaluation of the function of the vestibular nerve should be done in every suspected case of tumor of the eighth nerve or tumor involving the cerebellopontile angle.

The results of these tests give very valuable data and together with neurologic findings lead to a diagnosis.

It is sometimes wise to repeat the tests because the picture may change from time to time. The results obtained are due to intra-

cranial pressure, either local or general, and as this pressure changes so may the results of these tests change. This probably explains the difference in degree of involvement of the vestibular branches on the contralateral side found by different observers.

These tests are not disagreeable to the patient because they do not produce the dizziness and nausea experienced in the normal.

The following case illustrates the importance of the entire past history in evaluating the sequence of symptoms. Medullary symptoms, contralateral cranial nerve involvement and unusual cerebellar symptoms are likewise present.

CASE 2.—St. J. H., No. 7479-2 R 94-1929. Large right acoustic tumor. Right middle ear deafness, confusing chronology of symptoms. Partial enucleation. Death. Autopsy.

May 14, 1929, admission of Mrs. E. G., 38 years of age, a press operator, on the service of Dr. G. Neuhaus.

About three years ago she noticed a feeling of tightness or fullness in the right face, gradually extending to the left side of the face, now most marked over the nose and upper lip.

About a year ago she began to have occipital headache lasting about a week at the menstrual period only. At the same time there developed a constant discomfort behind the right ear.

For the past six months her vision has been failing. At times she sees double. A change of position, especially from lying down to sitting up, causes everything to turn backwards.

She has noticed for the past six months that she staggers from side to side when she walks, most markedly toward the right. For the same period she has had a buzzing in the right ear and the right side of her head. A right sided middle ear deafness dating from childhood has become worse for the past few months.

Recently she has noticed jerking of right arm and leg.

Positive Neurologic Findings: (a) General pressure. V., O. D. 15/50th; O. S. 15/50th. Fundus O. D. disc outline obliterated by swelling of five diopters. Marked hazy opacity of surrounding retina. Numerous hemorrhages about the disc. Left eye similar to right.

(b) Localizing. History of discomfort behind the right eye.

(1.) Cerebellar. Nystagmus to the left and spontaneous past pointing to the right with the right hand. Ataxia gait with staggering toward the right. Romberg towards the right. Adiodokokinesis on the right. Finger-nose test shows past pointing with the right hand.

(2.) Extracerebellar. Cerebral nerves. Fifth—subjective feeling of tightness and fullness over both sides of the face and now most marked over nose and upper lip. There is blunting of sensation of the right face. History that tongue feels too big. Sixth—negative, but history of diplopia. Seventh—cochlear branch. Air conduction is 0 on the right and bone conduction is decreased.

Vestibular branch. Bárány caloric test on right ear: Slight increase in nystagmus after three minutes. No vertigo after three minutes and no past pointing. The

same test on the left ear shows delayed reaction, but with vertigo and normal past pointing. Gives history of vertigo on changing position, especially from lying down to sitting up, when everything seems to turn backwards.

(3.) Medullary. Right biceps and triceps reflexes less active than left. Right patellar and ankle jerks less active than left.

(4.) Pupillary. Pupils equal and react to light, the right more actively than the left.

Clinical Diagnosis: Right cerebellopontile angle tumor. June 1, 1929. Operation—Left ventricle tapped through small incision over posterior horn. Cerebellar decompression, first stage. June 28, 1929. Removal of part of right cerebellopontile angle tumor.

Microscopic Findings: This tissue is basically fibrous tissue which occurs in large interlacing bands throughout most of the small section. In some places the bands are thin and cut longitudinally, while here and there they interlace with other bands which are cut crosswise. The bands vary in their cellularity, but in most areas the basic substance is wavy, moderately eosinophilic tissue, while the nuclei vary in number and in size and shape. Many nuclei are deep staining and tapering, while others are ovoid and large, moderately granular and contain single small nucleoli. There is a distinct palisading of the nuclei in areas, with closely packed nuclei arranged in parallel formation. The blood vessels are only venous and are rather widely scattered. The central portion of the small section is extensively vacuolated.

There is none of the reticular type of tissue commonly seen in this type of tumor.

Diagnosis: Acoustic neurinoma.

Subsequent Notes: After the operation she had difficulty in swallowing. She was quite restless at times. She continued to have difficulty in swallowing. On July 10, 1929, she became irrational and cyanosed and died July 15, 1929.

Postmortem Examination: "On removing the skull cap the dura is seen to be under considerable tension. The convolutions of the brain are flattened. A large bone flap has been removed from the occipital region and here the cerebellum bulges outward and is adherent to the scalp. In the right cerebellopontile angle there is a roughly spherical tumor nodule 3 cm. in diameter, causing a large depression in the right lobe of the cerebellum and the right side of the pons, and pushing both of these structures towards the left. The right eighth nerve can be identified at its origin and traced into the lower portion of the tumor capsule, where it disappears."

Microscopic: Sections of the tumor tissue reveal superficially the same type of tissue seen in the surgical tissue described above. The deeper portions of the specimen are composed of reticular tissue which is quite loose meshed and edematous. The reticular cells are in close relation with the reticulum and lie at the nodal points of the network. The surface of the tumor carries many good sized veins.

Diagnosis: Acoustic neurinoma.

Comment: When this patient first came for examination symptoms had been present for three years. The first symptoms indicated involvement of the right fifth nerve and soon there was indication of involvement of the left fifth. Any involvement of the right eighth nerve for the first two and one-half years after onset was obscured by the old middle ear disease on the right. Failure to properly evaluate this factor in the chronology of the onset of symptoms could have led to a misinterpretation of the tumor syndrome because of the dominance of symptoms

Fig. 2. Right pontile angle tumor from Case 2.

pointing to fifth nerve involvement. It could likewise lead to erroneous tumor localization or to a diagnosis of other extracerebellar tumors than acoustic neuromas.

In this case, too, we have contralateral cerebral nerve involvement, this time the fifth. This contralateral involvement has led in some cases to erroneous tumor localization.

Here, too, we find the medullary symptoms mentioned in the previous case, this time confined to a jerking of the right arm and leg.

The apparent rotation of self or objects are infrequent in Cushing's series and he does not believe that it is sufficiently common to be of diagnostic service. This patient presented one type of this rotation, as she stated that everything seemed to turn backwards when she changed from a lying to a sitting position.

The spontaneous nystagmus presented by this patient was towards the left. Cushing[1] feels that a nystagmus is of cerebellar rather than of vestibular origin.

The Bárány test showed somewhat different results in this case from the first case.

In the first case there were no responses from either vestibular nerve. In the second case a slight increase in the already present spontaneous nystagmus was elicited by stimulation of the vestibular nerve on the homolateral side. There was neither past pointing, vertigo nor nausea. Stimulation of the vestibular nerve of the contralateral side showed some responses but they were delayed.

The Bárány tests give accurate knowledge as to whether or not the vestibular branch of the eighth nerve is functioning. Personal

equation does not enter into these tests. While it is not conceded that the results obtained by stimulation of the vestibular nerve are reflex, yet they are not under the voluntary control of the patient. Therefore, when no responses occur following stimulation, one can be sure that there is a lesion either in the end organ or along the nerve pathway of sufficient size to paralyze the nerve.

These tests then are of diagnostic value in suspected lesions below the tentorium.

SUMMARY.

Two verified acoustic nerve tumors are presented to exemplify confusing medullary symptoms, contralateral cranial nerve involvement and other departures from the usual symptoms in these lesions.

536 CITY NATIONAL BANK BLDG.

BIBLIOGRAPHY.

1. Cushing, H.: Tumors of Nervus Acusticus. W. B. Saunders, 1917.

2. Henschen, F.: Zur Histologie and Pathogenese der Kleinhirnbrückenwinkeltumoren. Arch. f. Psychiat., 56:21, 1915.

3. Jones, I. H.: Equilibrium and Vertigo. J. B. Lippincott, Philadelphia, Pa., 1918, page 60.

XLVI.

THE SELECTIVE TREATMENT OF MALIGNANCY ABOUT THE HEAD.*

THOMAS C. GALLOWAY, M. D.,

EVANSTON, ILL.

The treatment of malignancy about the head has improved markedly within recent years. Perhaps the greater part of this progress has been in irradiation. Some of it has come from the employment of electrosurgery. Probably the most important factor, however, has been the better understanding of the relation of treatment to the tumor type, grade, location and other factors, and the more careful selection of the particular method for each case to give the best result. Though this must still be controversial, this discussion will attempt to define criteria for that selection of treatment.

The treatment of cancer is a many sided problem and it should not be approached from any single viewpoint. The physician who is committed to any one invariable measure is likely to give his patient much less than his best chance of cure. Cancer requires for its best result the combined knowledge and experience of all the men who can give important information.

That includes the pathologist who has studied tumors from the point of view of their reaction to irradiation, their natural course and their prognosis, the radiation therapeutist, and a surgically trained clinician. The latter should be capable of any exposure or operative procedure in his field, he should know how to use electrosurgery to the best advantage, he should have studied the various methods statistically and in individual cases and he must have the ability to preserve the judicial attitude in the face of improving technic. Any treatment which does not take into account the full potentialities of both irradiation and surgery (usually electrosurgery) is likely to fall far short of its goal.

In addition it is necessary to have abundant quantities of radium or high voltage x-ray, or both, and modern diathermy equipment.

*Presented before the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society at Indianapolis, January 23, 1935.

Given such a team and such equipment, any cancer about the head and neck, seen reasonably early, should have a fair chance for cure.

Though we may dream of a time when some general agent shall eradicate every cancer cell from the body—and some recent work is encouraging—for the present irradiation and electrosurgery dominate the field. Each has its advantages and disadvantages and each its proper sphere, which has been fairly well defined by increasing agreement among men who work with cancer. The division of the field will vary somewhat with the experience of the physician, with his ability to use either agent to its best advantage and with the type of his equipment or the quality and amount of irradiation available. No man lacking in either equipment or experience should proceed alone and the best results will be obtained in our field, I think, by a team of radiologist and laryngologist working with a competent pathologist.

The advantages of electrocoagulation, where it is applicable, have been repeatedly stressed.[1] They include rapidity and ease of operation, relative lack of hemorrhage, of shock and of postoperative pain and the sealing off of blood and lymph spaces against infection and metastases and it gives smooth healing. Tissue, malignant or benign, exposed to electrocoagulation is finally and irrevocably destroyed. It fails only because cancer cells are inaccessible to it or are in outlying strands or islands, or have already escaped to regional glands. It is destructive and necessarily sacrifices some sound tissue, usually a minimum of one centimeter about the growth. It has very definite operative risks, due chiefly to anesthesia, aspiration and hemorrhage, although these are far less than in scalpel surgery.

The advantages of irradiation are obvious. It does not terrify patients into avoiding treatment or temporizing. It usually leaves a good cosmetic result. It captures within its sphere of action outlying strands or early metastases that electrosurgery would miss. Its proper use is attended with little immediate risk.

Irradiation if improperly used has many bad effects. Insufficiently screened, it leads to destruction or very painful reactions of periostitis and perichondritis. Inadequately used, it may possibly stimulate cancer cells or seal them in scar tissue, hiding them until too late for surgery. It may by action on the cancer bed make the cancer radioresistant so that proper treatment later is without avail. It may so impair nutrition that later x-ray, radium or surgery causes terrific destruction and usually if the first treatment does not succeed it

fails—except as preoperative radiation may be followed by surgery at a short interval afterward.

It must be remembered that to be efficient either radium or x-ray must be used up to the maximum of normal tissue tolerance with a rather marked unpleasant or painful reaction, the so-called epithelite or epidermite and very definite and occasionally severe absorption effects.[2] It affects most, as a rule, the very tumors in which electrosurgery has fewest successes and these two agents, after all, make a very good team.

We cannot speak too dogmatically about radiation until we understand more specifically how it works. Even the recent report of the British Medical Research Council admits that the reaction to radiation is most complex and bewildering.

Regaud believes that action is on a susceptible nucleus; Ewing and Stewart think both nucleus and cytoplasm are equally affected. Other authors believe that a reactive fibrosis is the important factor and Pullinger believes that all effects of irradiation are due to vascular stimulation and vascular degeneration. There are probably two factors, early cancer cell necrosis and later destruction due to tissue reaction.

Very much has been written on radiosensitivity, and Stewart[3] required eighty-five pages to merely touch upon the high points of the subject.

He summarizes some of his conclusions as follows:

1. Sensitivity is only relative and ideas of tumor sensitivity will change with radiologic technic.

2. The effect of irradiation is a complex involving tumor cell, tissues of host and possibly general reactions.

3. Radiosensitivity may mean rapid tumor regression, progressive regression over days to weeks or slow atrophy of months to years.

4. Radiosensitivity increases with the degree of embryonal type and usually with anaplasia, but not all anaplastic tumors are radiosensitive.

5. Tumors are more radiosensitive in young, less so in old or cachectic or anemic.

6. Certain tumors are inherently resistant—e. g., melanomas and certain neurogenic tumors and desmoplastic tumors.

7. Normal tumor bed is favorable. Bone, fat and cartilage are unfavorable.

8. Infection interferes with irradiation.

9. Bulky tumors are resistant after infarction and liquefaction or with much secretion.

10. Metastases may vary from the parent, often are more resistant.

11. Rapid initial regression may lead to incomplete treatment, recurrence, radioresistance and failure.

The selection of proper treatment involves far more than histologic grading in given types, and the final judgment should be based on the gross appearance, clinical course, location and origin, previous treatment and local or general conditions of the host that help or hinder the best effect of radiation.

Yet the microscopic appearance, properly weighed, is of much importance. Definite types of tumor, as, for example, transitional cell carcinoma and so-called lympho-epithelioma, are very sensitive to radiation. Basal cell carcinomas are sensitive, as are round, reticulum-celled and lymphosarcomas. Adenocarcinomas and fibrosarcomas are relatively resistant.

Epidermoid carcinomas lend themselves to grading on the basis of relative maturity of cell types, abundance of stroma and other factors, and in Quick's[4] experience the gradings have been correct in the prediction of the expected action of radium within ten per cent error.

The degree of anaplasia is a rough indication of both malignancy and radiosensitivity, though lack of cell maturity, relative malignancy and radiosensitivity do not exactly parallel each other.

Melnick[5] and others have pointed out many possible errors in attempts at grading. Tumors of identical histologic appearance may run entirely different courses. Histologic criteria are not well established. Cell types are sometimes almost impossible to determine. Cells may be polymorphous. Mitotic figures may be inconstant, and there is growing evidence that amitosis or shortened form of mitosis may be important in tumor growth. In addition, as Melnick shows, a biopsy specimen may not be representative of the tumor as a whole; the character of the tumor may change with time, with blood supply, with irritation, infection, compression and other extraneous factors, and metastases may appear much different from the primary growth.

On the whole, however, histology may give valuable indications for treatment. Following the lead of Dr. R. Jaffe at Cook County Hospital, my clinical experience has been quite definite that cases with well matured cell forms, with much stroma, with hornification or other indication of specialization yield well to electrosurgery and often poorly to irradiation. Basal cell carcinoma, of course, gives good results with either. Very immature cell forms, undifferentiated, with little stroma, approaching grade IV of Broder's, yield much better to radiotherapy.

Location of tumors, in part at least due to the nature of the cells of origin, is an important factor in determining their treatment. The more radiosensitive tumors arise from the tonsillar regions, the pharynx and posterior third of the tongue. Favorable sites for electrosurgery are the skin of the face, ear, nose and lips, the maxillary sinuses, the palate, alveolar ridge, the cheek, the epiglottis and anterior third of the tongue.

The clinical appearance may be quite as important as other factors in determining treatment. If a fissured infiltrating tumor with poorly defined margins presents itself we can be doubtful about results with electrosurgery. If it involves in addition an unfavorable location, as the base of the tongue or lymphoid tissue of the pharynx, we may not waste our time and energy on it.

If it seems of short duration—though history is often to be doubted—and it seems to have grown rapidly, radiotherapy probably should be chosen. If there is much fixation and induration, except in long standing cases, it is probably not for electrosurgery. If the tumor has a well marked or rolled border, or is papillary or fungating, it is likely to be amenable to diathermy.

If regional glands appear early or are disproportionate to the original growth, the tumor is more likely to be of immature type and radiosensitive. The dissection of regional gland areas is of doubtful value in my experience and may actually remove an important barrier except that discrete unfixed masses left after radiation may well be removed. Preliminary radiation of glands and later interstitial implantation after reflecting the skin seem to give better results, with much less effort to patient and surgeon. It must be remembered, however, that metastases are frequently more radioresistant than the original growth.

We may say then if a tumor is of a relatively mature type, located in resistive tissue, of slow progress, of an appearance denoting

relative limitation, the major reliance may be upon electrosurgery. If just the opposite obtains, we should yield it willingly to irradiation and leave our energies for more likely material to the great improvement of our personal morale and of our statistics.

Preoperative x-ray or radium may be very valuable, both to the regional gland area and to the local lesion. It may also be valuable as a therapeutic test to determine the radiosensitivity. It may reduce the bulk of the tumor and its troublesome vascularity, as in the case of a hemangioma, a nasopharyngeal fibroma or chondrosarcoma. If it fails to completely eradicate the tumor, electrocoagulation can then finish the job.

Postoperative irradiation gives additional protection and should be done when there is any question of gland involvement or complete removal. If used it should be thorough.

General measures of importance include correction of asthenia and anemia, good nutrition, especially high vitamin diet and good mouth hygiene. We have recently seen a case with multiple skin carcinomas of three histologic types running wild brought under control by electrosurgery and high vitamin A diet.

The treatment of special regions may be briefly considered. Of the skin, basal cell carcinomas yield well to irradiation or coagulation or dessication with about 95 per cent cures or more if early. On the lids radium is probably better because it does not scar. Squamous celled cancers, I believe, are better coagulated, though irradiation, usually in caustic dose, will give 75 per cent cures in cases not too old. Combined treatment is excellent. For the lip, Pfahler's[6] statistics for electrosurgery followed by intensive fractional irradation seem as good as any.

Carcinoma of the antrum, even of an immature cell type, if still confined to the bony cavity, yields well to electrocoagulation, but exposure must be adequate and destruction radical. Preoperative and regional gland radiation and radium may be used in the cavity but must be very well screened to prevent painful periostitis.

If carcinoma of the tongue involves the base or is fissured or deeply ulcerating, radiation is the treatment of choice. If early, papillary and involving the anterior third or border, electrocoagulation will give good results, especially if combined with radiation. Electrosurgical removal of the whole tongue is safe, easy and not too mutilating, if radium fails locally, with glands uninvolved or controlled.

Carcinoma of the cheek will respond well to either endothermy or radium. If it involves the alveolar ridge or maxilla or mandible, coagulation, I think, should be used.

I have one five year cure of carcinoma of the tonsil by coagulation out of numerous attempts. On the other hand, Duffy could report 20 per cent and Coutard 28 per cent of five year cures in this region by irradiation, and these cases should surely be given over to x-ray or radium.

Carcinoma of the epiglottis and occasionally of the velum may be completely removed by electrocoagulation, but all others of the nasopharynx, pharynx and hypopharynx should be left to radium and x-ray. There is little justification, I think, at present for the formidable approaches to these tumors by an external route unless, as rarely happens, they are of a very mature cell type, and even then suspension and coagulation will usually give adequate exposure if they are operable.

The radiotherapists have discovered that x-ray or radium, well screened, using different ports of entry, may be used with relative safety in the presence of cartilage. With much enthusiasm, though the case does not yet seem statistically proven, some of them have seized upon carcinoma of the larynx as their own. The best figures for irradiation appear to be those of Coutard,[2] showing about 26 per cent of five year cures, although the seventeen cases of 1926, reported in 1931, gave 53 per cent cures, quite possibly showing a marked improvement in the technic. On the other hand, Jackson reports 82 per cent and Gluck-Sorenson 88 per cent of five year cures by laryngofissure in selected cases. St. Clair Thomson has pointed out that laryngofissure leaves a nearly normal individual with natural airway and serviceable voice. We should, I think, hesitate to turn such early cases to any more problematic method until time has proven its value.

Laryngectomy, I think, is still in more advanced intrinsic cases, with its 76 per cent cures given by MacKenty, to be preferred to radiotherapy, although it is easily possible that the latter may yet prove to be the method of choice. Radiation may give additional security as an adjunct postoperatively and is the choice, I think, in nearly all extrinsic cases. Coagulation is at times of value in cleaning up doubtful borders after laryngofissure and may at times be better than laryngectomy, if used through a thyrotomy after cartilage resection in cases which have overflowed the larynx above. The methods of Ledoux and Harmer or the method of Hautant and Coutard

with thyrotomy and mass destruction of the tumor, though incomplete, followed by irradiation or implantation, seems of much value, as shown by three cures in Coutard's statistics of cases of 1926.

Whether x-ray or radium is better is still debated by those specializing in their use. Hard Gamma rays reaching the depth of the tumor are the effective agent and can apparently be secured by either with proper filtration. Berven feels that the radium bomb is much superior, but Regaud believes both about equally effective. X-ray seems definitely cheaper. Radium is necessary for localized or interstitial use. To one who has observed their effect it appears that the use of either is dangerous if insufficient, because it makes tumors radioresistant, and if radium is used it must be well screened and used up to the point of maximum tolerance. If x-ray is employed it must be of high voltage—probably not under 200 kilovolts, given in fractional doses after the method of Coutard—well filtered, also to the point of greatest skin tolerance. Certainly there is no place for the careless, the casual, the inadequate use of either x-ray or radium.

This discussion has attempted to show that the otolaryngologist should not be committed to any one invariable treatment of malignancy. It has tried to define the best indications for the use of both irradiation and electrosurgery evolved by the co-operation of radiation therapeutist, pathologist and laryngologist.

636 CHURCH ST.

BIBLIOGRAPHY.

1. Galloway, Thomas C.: Malignancy About the Head. Arch. of Phys. Ther., 15:343 (June) 1934.

2. Coutard, Henri: Principles of X-ray Therapy of Malignant Diseases. Lancet, 2:1-8, 1934. Also Am. Jour. Rent., 28:313. (Sept.), 1932.

3. Stewart, F. W.: Radiosensitivity of Tumors. Arch. Surg., 27:979-1064. (Dec.), 1933.

4. Quick, D.: Practical Application of Radiosensitivity and Tumor Grading. N. Y. State Med. Jour., 33:1376-79. (Dec.), 1933.

5. Melnick, P. J.: Metastizing Leiomyoma of the Stomach. Am. J. Cancer, 16:18-22 (July) 1932.

6. Pfahler, G. E.: New Radiological Advances. Rad. Review and Chi. Med. Record, 56:83-88. (April), 1934.

7. Jackson, Chevalier: Cancer of the Larynx: Its Curability by Laryngofissure. S. G. O., 58:431. (Feb.), 1934.

8. Berven, E. G.: Development of Technique and Results in Treatment of Tumors of Oral and Nasal Cavities. Am. J. Roentg. Rad. Ther., 28:332-342. (Sept.), 1932.

XLVII

PATHWAYS OF APPROACH TO THE PETROUS PYRAMID.*

MARVIN F. JONES, M. D.,

NEW YORK.

Avenues of approach to the petrous pyramid have been the subject of many discussions and experiments, yet in the light of present knowledge it seems probable that several approaches must be employed. These will depend upon the anatomic pathways of invasion, and upon the resultant courses of infection. The anatomic pathways have been ably enumerated by Profant,[1] Meltzer,[2] Friesner and Druss[3] and others. The term "petrousitis," which was proposed by Profant,[1] signifies an inflammation in the petrous portion of the temporal bone. This term will be used to avoid the confusion of qualifying terms, such as osteitis, osteomyelitis, empyema of the petrous tip, etc. A few procedures for surgical relief have ben proposed, but no method yet advanced is applicable to all cases. It is my belief that the petrous process is subject to the same types of involvement as the mastoid process. The mastoid is subject to an erosive process without demonstrable pus, and it seems a reasonable theory that the petrous process may be likewise involved. I rather hesitate to propose this assumption since it is based on a single case. During the exploration of a mastoid for a causative focus of a leptomeningitis, I opened the petrous process through its roof. There was a foul odor similar to that which is noticed when a cholesteatomatous cavity is opened in the mastoid process. No pus was found. We all recognize the acute purulent petrousitis, and I believe nonpurulent petrousitis deserves consideration.

The methods of invasion of the petrous pyramid have been fully discussed. The most logical avenues seem to be through the cells contiguous to and continuous with those of the mastoid process and the middle ear. The theory of infection progressing along the blood, lymph channels or nerves is logical. The erosive type of petrousitis progresses through diploic structures, thus reaching a nonpneuma-

*This paper was presented before the Otolaryngological Section of the American College of Surgeons at its meeting in Boston on October 19, 1934.

The histologic specimens presented in this paper have been made possible through a grant by the Central Bureau of Research of the American Otological Society.

Fig. 1. A vertical section through the eustachian tube of a temporal bone demonstrating the hypotympanic cells and the peritubal cells going forward around the bend of the carotid artery under the cochlea to the petrous tip.

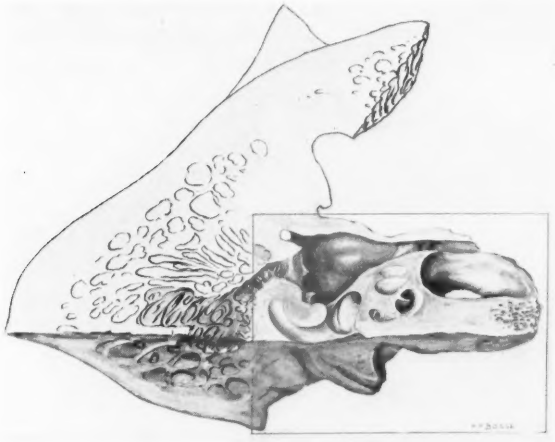


Fig. 2. Temporal bone cut in two planes viewed from above, demonstrating an almost complete sclerosis of the petrous pyramid and the relation between the carotid canal, the cochlea and the eustachian tube.

tized petrous tip. A superimposed purulent process is afforded an easy access to devitalized areas, and the extension through a degenerated petrous cortex would be a natural result. The occurrence of osteomyelitis in the temporal bone similar to the process occurring in the frontal bone is rare. The petrous pyramid is the occasional seat of a true osteomyelitis.

A most comprehensive survey relating to the subject of "petrousitis" was published in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY in June, 1926. The title is "Otogenic Paralysis of the Abducens," and the author is William Hardin Sears of Huntingdon, Pa. The subject was presented as a candidate's thesis for the American Laryngological, Rhinological and Otological Society. I am purposely giving this reference prominence because it correlated the rather scattered data published previous to 1926, and was the precursor of a rapid advance in the diagnosis and surgical relief of petrousitis.

The symptomatology of this disease has been limited until recently to the manifestations of an advanced stage, the Gradenigo symptom complex being the traditionally accepted triad of diagnostic value. Kopetzky and Almour[4] stress the orbital pain feature. This is a valuable contribution in that it may indicate the location of pus at an earlier stage. Vail[5] has contributed an engaging and plausible explanation of the orbital pain, and another localizing symptom of an earlier stage may well be the vestibular attacks observed by Bowers.[6] Impaired hearing may be added. I have observed sudden impairment of hearing in some cases, yet in reviewing the literature I find no emphasis placed on this symptom. The vestibular symptoms are logical indications of petrousitis, and we have the term perilabyrinthitis to indicate such an involvement. Two of my cases have presented the perilabyrinthine symptoms. The variety of symptoms which may indicate the progression and location of earlier involvements are indicated by Sears in his enumeration of the pain elements. The pain was localized as frontal, temporal, parietal, facial, auricular, occipital, neck, orbital and periorbital. These pains were characterized as "agonizing," "unbearable," "intense" and "severe." Persistent discharge from the canal or mastoid wound should make us suspicious of petrousitis. Profant has mentioned this feature, and we have all encountered cases of mastoiditis which continue to discharge a distressing amount of pus, in spite of any and all accepted operative procedures. Various explanations have been offered, most of which were as unsatisfactory as the operative results. The theory that pus may be an indication of infection extending into the petrous process

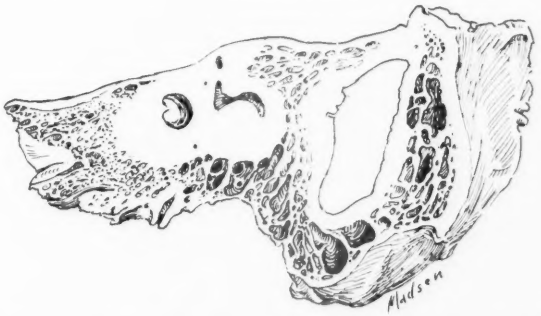


Fig. 3. Demonstrating the hypotympanic cells extending forward under the labyrinth with a transition from the pneumatized type to the diploic type.

Fig. 4. Demonstrating the extension of the hypotympanic cells, forward towards the petrous tip with special attention being called to one long continuous cell in the posterior superior region of the petrous pyramid which extends continuously forward to the petrous tip.

is at least worthy of consideration. A persistent, uncontrollable aural discharge should lead to a minute investigation of its origin.

It is obvious that the stage of progression plus the location and extent of involvement assume major proportions in determining the method of surgical attack. Two principal methods of approach have been proposed. Eagleton[7] advocates an entrance over the anterosuperior and posterior surface of the petrous process. Kopetzy and Almour[8] advocate an entrance to the infected area through the body of the petrous. Both methods have produced favorable results in the hands of their advocates. Another approach is founded on the procedure of Streit,[9] and has been successfully employed by Lilly[10] and Seydell.[11] This method is actually a considerably modified combination of the two above mentioned procedures. Other variations of the Eagleton approach have been advocated and performed successfully by Smith[12] and Myerson.[13]

The various methods may be summed up as follows: An entrance is gained to the cranial cavity by removal of the temporal squama. Through this opening an exploration of the anterosuperior surface of the petrous process is possible. This may be termed the external approach. The second method advocates transfixing the body of the petrous process with a dental drill which passes in the angle formed by the carotid artery and the whorls of the cochlea. Both these procedures necessitate a preliminary radical mastoid operation. The third is really a combination of the first two in that it follows the course of cells transfixed by the drill and at the same time removes the petrous cortex or floor of the middle fossa. Seydell claims a preliminary radical mastoidectomy is not always necessary. These three procedures aim at the superior group of cells. In order to reach the posteromesial surface of the petrous process an external approach is necessary. The dura over the superior posterior edge of the petrous process is elevated, thereby gaining entrance to the posterior cranial fossa.

My own experience with one of these methods will demonstrate a pitfall. I had carefully surveyed a number of temporal bones and felt that I had determined mathematically an exact approach. I used the superior semicircular canal as a landmark, and I chose the Eagleton as a basic method because it appeared less dangerous, although more formidable, than the Almour. Using this method, I had succeeded in entering the superior cells. This work was done on the cadaver. I felt so elated by my success that I gave a demonstration of the procedure before the Otolaryngological section of the New York Academy of Medicine. Instead of entering the petrous cells as

Fig. 5 (Same specimen as Fig. 4). Demonstrating the extension of the hypotympanic cells forward towards the petrous tip, with special attention being called to one long continuous cell in the posterior superior region of the petrous pyramid which extends continuously forward to the petrous tip.

Fig. 6. Demonstrating the posterior superior ridge of the petrous pyramid with an over-hang which is not uncommon, making an approach to the posterior surface of the petrous pyramid a physical impossibility.

I had planned, I plunged my curette into the carotid artery. Anatomic anomalies do occur, and fortunately I had found my first one on a cadaver. These same anatomic variations are probably the greatest hazard for any safe surgical approach.

In cases where pus has escaped the confines of the petrous process and collected outside the dura in the middle fossa, any approach over the surface of the petrous has produced satisfactory results. Collections of pus in the posterior fossa are less easy to reach and more difficult to drain. The technic devised by Almour and advocated by Kopetzky impressed me, as a casual observer, as being extremely dangerous. Being interested in their method, I had rehearsals with the cadaver, as is my custom in trying new operative procedures. I was surprised at the ease and exactitude with which the superior cells were entered. I must admit one failure. A particularly well pneumatized mastoid was selected. The petrous portion, however, proved to be nonpneumatized, and my drill went through the cochlea. A fraction of a millimeter anterior would have opened the carotid. The distance traversed by the drill is surprisingly short, 3 to 5 mm. (Almour); yet the leeway obtained by a proper preparatory operation is ample for comparative safety if the petrous is pneumatic. The Kopetzy-Almour approach is indicated when their syndrome denotes involvement of the superior cells. The results they have obtained are sufficient to warrant further trials.

There is another approach deserving development; one which would drain the cells in the dependent portion of the petrous pyramid, where drainage is aided by gravity. The area thus drained might not be reached by any of the aforementioned methods. It is by way of the hypotympanic cells. Our dissections and histologic specimens show that these cells are commonly present and are of a large caliber. Their extent is varied, and that they should be frequently involved is easily understood when their proximity to the middle ear is considered. Mayer[14] mentioned drainage through these cells, and employed the method successfully. Shea[15] made mention of the same route; Haskin suggested its possibilities before the New York Otological Society. Bowers used the method successfully on a single case, and I have employed this approach with success in one case. The radiogram taken will be shown by the lantern slides.

The technic was simple: The middle ear cavity was thoroughly cleaned and then anesthetized with a 10 per cent cocain and a 1-10,000 adrenalin solution. The fundus was examined by a magnifying otoscope, and several small depressions presented themselves in the hypotympanic and tubal areas. These were explored with a fine, blunt,

Fig. 7. Showing the normal side of a petrous abscess case. The pyramid shows the usual cell formation on the non-affected side.

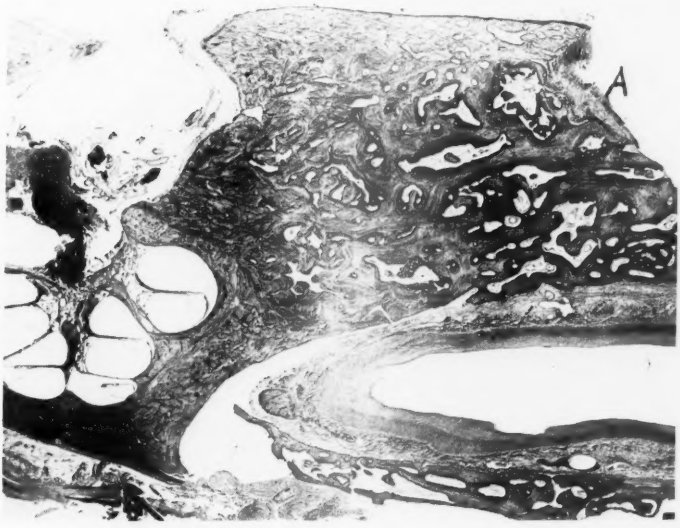


Fig. 8. Shows the sclerosed bone in the petrous pyramid on the diseased side of the same case represented by Fig. 7. The abscessed cavity which caused excavation of the bone is labeled "A".

Fig. 9. Sketch from the actual specimen showing the excavated bone in the floor of the middle fossa mesial and lateral to the superior and semicircular canal running forward in the petrous pyramid. The carotid artery is also illustrated, showing its vulnerable position in any procedure which explores the petrous tip over the superior surface of the petrous bone.

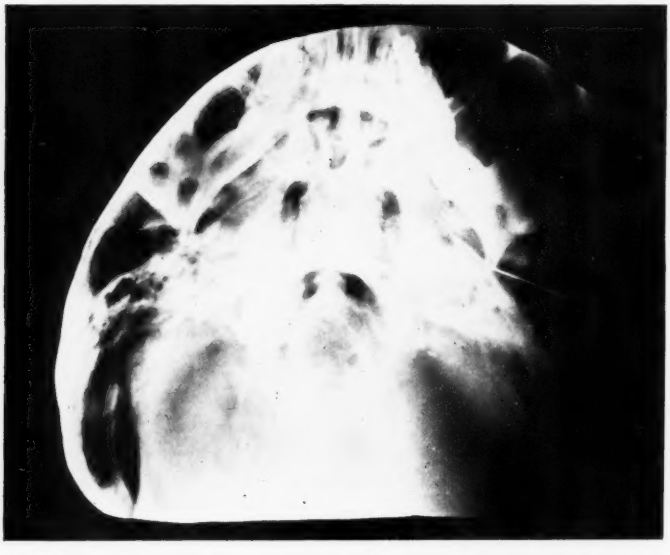


Fig. 10. Silver probe in petrous pyramid of case No. 6. (B. F.)

silver probe. Finally, the probe entered a cell and was gently advanced until its progress was terminated by solid bone. The distance will be shown by the roentgenogram. Following the removal of the probe, pus welled out; and the patient was relieved of a deep seated parietal pain from which she had suffered for over a year. Her dizzy attacks also ceased. She had one recurrence of symptoms which was relieved by enlarging the opening of the sinus. To date she is symptom free. If the sinus has formed, be it in the middle ear, mastoid process or one artificially created, drainage produced by enlarging the opening or cleaning away granulations seems to be adequate.

Most otologists observe indications of petrousitis, and the general feeling is that most cases will resolve following adequate surgical treatment of the mastoid. This is undoubtedly correct in acute cases. The acute exacerbation of a subacute case must be carefully watched for any adverse manifestations, and cases with a history of long duration are especially alarming because of the rapid onset of symptoms indicating intracranial involvement. The condition preceding these grave symptoms is usually one of a benign purulent otitis.

The subject of petrousitis is a fascinating topic to study. Additional pathways of approach will have to be devised and existing methods improved in order to drain the various areas involved. There is also much work to be done in the field of cataloging the premonitory symptoms which will indicate the extent and location of a progressive destruction.

REPORT OF CASES.

CASE No. 1. (F. H.)

Complaint: Falls to the left; severe pain around the left ear; purulent discharge; rotation of external objects; nausea and vomiting.

Examination: Rotatory nystagmus on looking to the right and upward. Irrigation of the left ear did not change the nystagmus. Heard a spoken word at 10 feet with a noise apparatus in the right ear. Optic disc, edges blurred. Spinal fluid: under normal pressure.

Operation: Operation performed with a relief of symptoms (severe left headache).

CASE No. 2. (M. Z.)

Complaint: Headaches, dizziness, nausea, aural discharge, tinnitus, deafness.

Examination: Horizontal nystagmus to the right. Rhomberg backwards and to the right. Spontaneous pastpoint to the left. *Fundi:* Discs brownish red. Nasal margins indistinct. Some sclerosis on both sides. Caloric test (water at 68 deg.), produced no reaction after four minutes. Kobiac test showed the vertical canals to be functioning, and no response to the horizontal.

Operation: Operation performed with a relief of symptoms.

CASE No. 3. (M. M.)

Complaint: Impaired hearing, tinnitus, left-sided headache, dizziness, nausea and vomiting.

Examination: Nystagmus to the right, discharging left ear. Caloric test showed the labyrinth to be functioning.

Operation: Operation performed with complete relief of symptoms.

CASE No. 4. (J. S.)

Complaint: Aural discharge from the right ear, vertigo, pain in the right side of face; hemicrania so severe on the right side as to prevent sleep; heavy feeling in the right eye; impaired hearing.

Operation: Operation performed with relief of symptoms.

CASE No. 5. (A. S.)

Complaint: Ringing in the right ear, discharge, dizziness, very severe headache, nausea; falls to the left; impaired hearing.

Examination: Nystagmus to both sides, more to the right than to the left; spastic hemiplegia since birth; external strabismus, right; primary optic atrophy.

Operation: Operation performed with relief of all symptoms except dizziness.

CASE No. 6. (B. F.)

Complaint: Ringing in the ear, deep-seated pain, discharge, dizziness, vertigo, nausea, falling to the left, impaired hearing.

Examination: Nystagmus, both sides; more marked to the right than to the left; falling to the left; difficulty in locomotion.

Operation: This is the case described in the body of the paper in which the probe was directed into the petrous pyramid, resulting in an overflow of pus with the relief of symptoms.

IN THE SERIES OF SIX CASES.

Complaint	*Number*
Chronic aural discharge	6
Lateralized headaches	6
Persistent dizziness or dizzy attacks	6
Impaired hearing of varied degrees	6
Nausea	5
Tinnitus	4
Unable to sleep because of pain	3
Vomiting	2
Retro-orbital discomfort	1

Examination	*Number*
Nystagmus	5
Falling	4
Disc changes	3
Pathways of infection into the petrous pyramid was demonstrated	3
Pathways of the eustachian tubes found obliterated	2
No response to the horizontal canal on the infected side	1
No labyrinthian response	1
Disturbances in locomotion	3

The case reports were presented before the monthly conference at the Manhattan Eye, Ear and Throat Hospital in December, 1934.

The operative procedure employed in the cases I have reported is based on the same broad principle which is followed in all mastoid surgery. This procedure consists in eradicating cells wherever they may be found. A preliminary extensive radical mastoidectomy is done, and broad exposure of middle fossa dura is accomplished by a wide removal of the squamous portion of the temporal bone. Cells lateral to the superior semicircular canal are removed by curette and rongeur following the floor of the middle fossa forward and mesialward. Cells mesial to the superior semicircular canal are eradicated by the same method, and likewise hypotympanic and peritubal cells are curetted cautiously. The subarcuate area is explored by a small silver probe, as are all the other exposed cellular areas, and a sinus tract leading forward into the petrous pyramid may thus be demonstrated. It had been my custom to elevate the dura over the superior surface of the petrous pyramid, but recent experience had taught me that this part of the technic is unnecessarily hazardous. If an extradural abscess is suspected, most of the elevation will have been accomplished by the pathologic process. As previously pointed out, and as must be obvious, this procedure has its pitfalls; yet from my experience and observation, it is the least hazardous and has been successful in the six cases herein reported.

I wish to express my thanks to Dr. Frederick A. Hemsath, whose opinions as pathologist have been of great assistance to me in preparing this work.

121 EAST 60TH ST.

BIBLIOGRAPHY.

1. Profant, H. J.: Gradenigo's Syndrome with a Consideration of Petrousitis. Arch. of Otolaryng., 13:347 (March), 1931.

2. Meltzer, Philip E.: Gradenigo's Syndrome: Anatomic Aspects. Arch. of Otolaryng., 13:87-93 (Jan.), 1931.

3. Friesner, I., and Druss, J. G.: Osteitis of the Petrous Pyramid of the Temporal Bone. Arch. of Otolaryng., 12:343 (Sept.), 1930.

4. Kopetzky, Samuel J., and Almour, Ralph: The Suppuration of the Petrous Pyramid. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 40:137 (Mar.), 1931.

5. Vail, Harris H.: Vidian Neuralgia with Special Reference to the Eye and Orbital Pain in Suppuration of the Petrous Apex. Arch. of Otolaryng., 17:212-221 (Feb.), 1933.

6. Bowers, Wesley, O.: Two Cases of Petrous Abscesses: Recovery. Laryngoscope, 38:415 (June), 1928.

7. Eagleton, Wells P.: Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex. Arch. of Otolaryng., 13:386-442 (Mar.), 1931.

8. Kopetzy, Samuel J., and Almour, Ralph: The Suppuration of the Petrous Pyramid. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 40:397-414 (June), 1931.

9. Streit, H.: Ein Operationsverfahren zur Eroffnung Tiefgeleggener von der Spitze der Felsenbeinpyramide aus Gehender Epidural Abscesse. Arch. f. Ohrenheilk., 57:169 (1902-1903).

10. Lillie, Harold, and Williams, Henry L.: Suppuration of the Petrous Pyramid. Arch. of Otolaryng., 15:693 (May), 1932.

11. Seydell, Ernest M.: Suppuration of the Petrous Pyramid. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 42:462 (June), 1933.

12. Smith, C. H.: Epidural Abscess Over the Tip of the Pyramid with Symptoms of the Fifth and Sixth Nerves. Arch. of Otolaryng., 14, 337-338 (Sept.), 1931.

13. Myerson, M. C., Rubin, H. W., and Gilbert, J. G.: Improved Operative Technic for Suppuration of the Petrous Apex. Arch. of Otolaryng., 19:699-703 (June), 1934.

14. Mayer, Otto: Gradenogscher Symptomkomplex bei Arkuter Mittelohreiterung. Monatschr. f. Ohrenh., 63:970, 1929.

15. Shea, J. J., and South, M. J.: Petrous Tip Cells a Projection of the Hypotympanic Cells.

16. Sears, Wm. Hardin: Otogenic Paralysis of the Abducens. Candidate's thesis for the American Laryngological, Rhinological and Otological Society. (June, 1926, published in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY.)

XLVIII

PHONIATRY IN DYSPHONIA VENTRICULARIS.*

CHARLES H. VOELKER, M. A.,

COLUMBUS, OHIO.

There is a type of unpleasant vocal quality which is characterized by a rattling, rumbling, cracking or ticker-like substitution for phonation. This timbre may resemble more a noise, but again, may have considerable regularity. It may vary from about six to thirty, or more, vibrations per second. The physiology of production indicates that this phonopathy could be called dysphonia ventricularis. The false vocal cords, or ventricular bands, supply the vibration rather than the true vocal lips.

The logophonopathology seems to be entirely one of voice quality. The vowel quality is completely understandable. All phonetic sounds are easily produced by vibration of the ventricular folds. The significance of this phenomenon, in regard to the function of the glottis in vowel quality, is not within the province of this paper.

In certain ordinary situations this peculiar vibratory initiation appears. It seems to be an alternate for pneumothorasad phonic utterance. In regard to the use of inspirative pronunciations, it is rather common to hear inspirative voice production on such words as "yes" and "no" when the one replying is conversationally distracted and concentrating upon some ulterior thing. However, it is also common experience in this situation to hear either an inspirative or an expirative ventricular phonation substitution. The difference between the inspirative and expirative ventricular vibration is not nearly so pronounced as it is with the glottal orad and thorasad phonation. All types, as well as many other idiophonias, are commonly used in uttering certain exclamations.

A considerable percentage of some of my classes have demonstrated this vocal unpleasantness, dysphonia ventricularis. What the students specifically did was to complete sentences by speaking in a careless and indecisive manner. The result, in some cases, was the ventricular substitution for phonation. The phoniatric procedure was simply a matter of establishing a criterion for fineness of enun-

*From the Department of Speech Pathology, Capital College, Columbus, Ohio.

ciation in the minds of the students. Any ordinary principles for excellence of speaking or reading proved applicable. The speech re-education revolved around decisiveness of speaking.

One case, a college student, had developed this idiophonia in high school as a result of overcompensation for physical inferiority. It was an acquired part of his personality and, as a result, merely intermittent. In this case the therapy consisted in developing the original voice into a mellow, vibrant quality, superimposed upon forceful intonation. The result was more pleasant than the false voice, besides negotiating an actual alteration in the personality of a definitely superior character, either to that portrayed in the assumed quality, or to the original timbre.

Another case, which demonstrated this phonopathy, might be classified as aphonia ventricularis, if the Greek stem indicates solely glottal phonation, as, in all probability, is the present significance. This case, a student French teacher, in no situation produced a vibration of the vocal cords in speaking, only a ventricular vibratory substitution. Physiologically her laryngeal mechanism was unimpaired. The etiology of the voice defect, however, was not disclosed. Nevertheless, it might be suggested that it could have been socio-environmental in origin. Her intonation and phonetic pronunciations were unimpaired. However, there was a tinge of quality in the delivery which resembled a mild paraphonia athymica.

In treating dysphonia or aphonia ventricularis, any attempt at tensing the gross myology in order to procure a sympathetic laryngeal muscular contraction of such a nature as to draw the vocal bands together, is fruitless. The strong mutual, physiologic relationship between the tensing of the laryngeal sphincter and gross kinetic contractions defeats any such therapy. It also seems to be useless to employ an induction current as is sometimes used in aphonia hysterica. In other words, the seat of this phonopathy lies not in the glottal action, but in the constant employment of the laryngeal sphincter for articulation. Any therapeutic procedure in such speech re-education must endeavor to relax this sphincter.

The phoniatric procedure is quite simple, but must proceed cautiously. First, the patient should be trained to extend the superior laryngeal aperture. The best means for this is to open the mouth and take a full inspiration of breath, as in yawning. Incipient pandiculation should be practiced as an exercise until this vocal key position can be assumed with facility and will not progress into the uncontrollable stages of the complete pandiculation. Second, maintaining

a relaxed laryngeal sphincter, the patient should practice respiratory gymnastics. Then, likewise retaining the principles of procedures one and two, the patient should practice whispering the vowel sounds. And finally, the patient must practice vowel phonation after this manner, gradually building up ability through consonants, syllables and words, until connected speaking becomes a possibility.

1452 North High Street.

XLIX.

LAROCAINE AS A LOCAL ANESTHETIC FOR THE NOSE, THROAT AND ENDOBRONCHIAL SURGERY.

HOWARD L. STITT, M. D.,

CINCINNATI.

To Koller, the Viennese oculist, who, in 1884, announced to members of the German Ophthalmologic Society that he had discovered the anesthetizing properties of cocain, must go the credit for opening that new era in surgery made possible through perfecting methods for local, regional, surface, infiltration, sacral, spinal anesthesias. A quarter of a century marks the time elapsing between the isolation of the alkaloid from the leaves of erythroxylon coca by Niemann and its clinical application by Koller, who demonstrated to his colleagues that the drug was effective, whether it be administered by injection or application to mucous membranes. Publication of Koller's observations resulted in surgeons the world over adopting the new method of anesthesia. This was particularly so in the fields of otorhinolaryngology, ophthalmology and orthodontia. As early as 1884, Kelly, who successively occupied the chair of gynecology at the University of Pennsylvania and Johns Hopkins, applied cocain for inducing anesthesia. In 1885, Halsted of New York employed the drug for infiltration anesthesia, and during the same year Corning demonstrated its effectiveness in spinal anesthesia.

Cocain, unfortunately, is not a very safe drug. Procain is of no utility for surface anesthesia, as it is not absorbed to any great extent from the mucous membranes of the nose, throat, larynx and bronchi.

We have diligently sought a substitute which would act as well as cocain in inducing surface anesthesia and yet be relatively non-toxic, non-habit forming and non-narcotic. We have not been disappointed in our search.

Nearly three years ago our attention was directed to a new surface anesthetic which had recently been discovered by Mannich. The drug has been introduced into the materia medica as larocaine. Chemically it is the hydrochloride of paraeminobenzoyl—2, 2—dimethyl, 3—dimethylamino, 1—propanol. It occurs as a white, crystalline powder which melts at 197 degrees C. It is easily soluble in water, less so in alcohol.

Larocaine belongs to the series of anesthetics which are obtained by the esterification of an amino-alcohol with para-aminobenzoic acid. Its demonstrated advantages over similar compounds are said to be due to the favorable arrangement of the acyl- and amino-groups in the molecule. This agrees with the principle laid down by von Braun that the best physiologic action is shown by compounds in which the amino- and esterfied hydroxyl- groups, separated by three carbon atoms, are in the alpha, gamma position. The structure of larocaine is also characterized by an acylated primary alcohol group and a quaternary carbon atom in the beta position, and which does not bear a replaceable hydrogen atom. Aqueous solutions of the drug can be sterilized by boiling for ten minutes. Stock solutions are to be kept in dark bottles. Epinephrin is added to larocaine solution just prior to administration.

Various workers have studied larocaine pharmacodynamically and published reports of these animal experimentations indicate the fine results which one may expect when this new cocain substitute is used in clinical application. Hirschfelder and Bieter, in reporting their study of local anesthetics, point out that larocaine is somewhat less toxic than cocain, but on the rabbit's cornea produces anesthesia in a lower concentration and much longer duration. Larocaine is non-irritant and, compared to procain, it is anesthetic to the sensory nerve fibers in the frog in one-half concentration of procain.

Fromherz gives the lethal dose of larocaine for the mouse as 0.3 gm. per kilo by subcutaneous injection, whereas the lethal dose of cocain is 0.1 gm. In the rabbit, the lethal dose of larocaine is 0.15 gm. per kilo, compared with 0.07 gm. of cocain. Lethal doses produce respiratory failure which is easily overcome with artificial respiration. In the rabbit respiration and in the cat, even heart action may be restored after complete arrest. Larocaine is rapidly detoxified in the organism and therefore any action on respiration or circulation from therapeutic doses could be of only the briefest duration. It exerts no irritant action on the cornea, even in solutions of from 10 to 20 per cent concentration. Administration of larocaine by injection does not produce any tissue inflammation or destruction. In vitro, it produces hemolysis in concentrations of 1:60, while procain does so in concentrations of 1:40. In concentration of 1:100 larocaine does not produce hemolysis within ten hours; in therapeutic doses the hemolytic action is nil.

Dietrichs, in his studies found that larocaine, in the concentration of 0.08 per cent, abolishes the conduction capacity of the

sensory nerve and those of the motor nerve in a concentration of 0.085 per cent. In a concentration of 0.125 per cent it produces a corneal anesthesia upon the eye of the rabbit and causes a "quaddel" anesthesia in 0.03 per cent concentration. Combining larocaine with epinephrin results in prolonging the effect of the anesthetic.

Numerous reports have appeared in the literature concerning the use of larocaine in clinical application. Among these are many attesting its effectiveness in the field of laryngology, otology and rhinology. Koch tells us that in ophthalmology, after rejecting other cocain substitutes, he finds a five per cent larocaine solution as effective as a ten per cent cocain solution. He also finds that its action is rapid, more powerful than cocain, and finally less irritant than other cocain substitutes which he has used. Hamburger is able to initiate minor nasal interferences after spraying each side of the nose for a few minutes with 10 to 15 cc. of a 0.5 per cent larocaine solution. Hopstein use 10 to 20 per cent larocaine solution for surface anesthesia, and a 1 per cent solution for infiltration anesthesia. He reports 171 cases which comprise Caldwell-Luc operations on maxillary sinuses, endonasal openings, submucous resection of septum, clipping of hypertrophied turbinated bone, adenectomy, incision of peritonsillar abscess, tonsillectomy, paracentesis of maxillary sinus.

Jent cites a case of severe aphthous stomatitis with difficulty in swallowing in which a larocaine gargle proved beneficial. Petermann uses larocaine, in 5 per cent concentration, for surface anesthesia, and a 1 per cent solution for infiltration anesthesia. He reports its use in seventy cases which include punctures of antrum of Highmore, esophagoscopies, frontal sinus operations and ethmoid scraping.

Ridpath desensitizes the epiglottic with larocaine in place of cocain and relieves the congestion without any systemic effect of secondary congestion. For surface anesthesia, Schoenlank employs a solution containing larocaine 2.5 per cent; sodium chloride, 0.9 per cent, and potassium sulphate, 0.4 per cent. For anesthetizing the mouth and larynx he uses the solution with the addition of epinephrin; for use before the cautery, excision of small tumors, and for diagnostic procedures he uses 30 to 45 drops of the solution to which are added 6 to 10 drops of 1:1000 epinephrin solution.

Taegen employs larocaine for anesthetizing the ear drum. Vock reports the use of larocaine for anesthetizing nasal mucosa in seventy cases. In a later paper he tells of forty-five examinations of the esophagus and forty-five of the respiratory tract, particularly in

cases for removal of a foreign body or for exploratory incision, which were carried out following spray or instillation of a 10 per cent larocaine solution. His cases also include fifteen bronchoscopies and fifteen esophagoscopies with 5 per cent larocaine solution instilled into the larynx or esophagus for purely diagnostic purposes.

In our own cases, which now number several hundred, we have found larocaine to be a very satisfactory surface anesthetic and one capable of replacing cocain for all routine uses in the nose, throat, trachea and bronchi. We employ the identical techic as we did with cocain and to date have not observed the slightest symptom of toxicity.

Due to the hypertonicity of the liquid, the irrigation of nasal passages with Bledsoe-Fisher solution is attended by quite some pain. It has been found that such discomfort can best be avoided by the application of a 3 to 5 per cent solution of larocaine to which a small quantity of epinephrin is added. This is applied, on small cotton pledgets, deep into the middle meatus, and allowed to remain there from three to five minutes, at which time the Bledsoe-Fisher solution is administered. We have had several hundred such cases. No shrinkage of the mucous membranes has been observed, and the resulting anesthesia has been as satisfactory and also of longer duration than that induced by cocain. A 5 to 10 per cent larocaine solution is of sufficient concentration for anesthetizing the pharynx and larynx when used as a spray.

By far the most important use to which we have put larocaine has been for inducing anesthesia in bronchoscopies and for bronchial lavage with the Coudé catheter. We have done twenty bronchoscopies under larocaine anesthesia and our bronchial lavage cases number more than 150. A 10 per cent solution is used to spray the larynx and a 5 per cent concentration for intracheal injection. For the trachea, not more than 15 cc. of the 10 per cent, or at times 5 per cent larocaine solution are required to obtain a satisfactory anesthesia.

It has been our observation that a 10 per cent larocaine solution, on a cotton pledget beneath the inferior turbinate, to induce anesthesia before a sharp needle puncture of the maxillary antrum is as effective as cocain while its action is of longer duration. A 10 per cent solution induces sufficient anesthesia for lavage of the maxillary antrum through the natural opening.

We also successfully carried out anesthetization of the sphenopalatine ganglion by placing over the area a cotton tip probe saturated with a 10 per cent larocaine solution. In such cases the neuralgic

pain is promptly relieved and the effect of the drug more lasting than cocain, in identical concentration, had been in similar instances.

At this time it should be pointed out that in our work the concentrations of the larocaine solutions have been identical with those of the cocain solutions formerly used. It has been found that the effect produced by larocaine is more prolonged than that induced by cocain. In our experience, toxic and other untoward by-effects, such as headache and giddiness, so common with cocain, have not been observed following application of larocaine. In fact, our experience with the drug has been so favorable that we now furnish tonsil patients with a 5 per cent solution of larocaine to be sprayed from an atomizer for the control of postoperative pain. No deleterious effects or delayed healing have been observed.

SUMMARY.

In clinical application as a surface anesthetic in several hundred cases larocaine proved as effective as cocain, with the resulting anesthesia being of longer duration. Toxicity or other by-effects have never been observed. We find that larocaine is capable of replacing cocain for all routine uses in the nose, throat, trachea and bronchi. Our experiences with the drug confirm the observations of other clinicians, especially Vock, who recommends larocaine as a most suitable anesthetic in bronchoscopic work.

19 GARFIELD PLACE.

BIBLIOGRAPHY.

Hirschfelder and Bieter: Local Anesthetics. Physiological Reviews, 12:190, 1932.

Fromherz: Locain, ein neues Lokalanaesthetikum. Arch. f. exper. Path. u. Pharmak., 158:368, 1930.

Dietriches: Die Lokalanaesthetishen Eigenschaften des p-amino-dimethyl-amino-propanol-benzoyl-ester (Larocaine). Arch. f. exper. Path. u. Pharmak., 161:206, 1931.

Koch: Kurze Mitteilungen ueber Klinische Versuche mit Kokainersatmitteln. Deutsch. med. Wochenschrift, 57:678, 1931. Larokain Roche' ein vollwertiges Oberflaeschenanaesthetikum. Arch. f. Ohren., Nasen- und Kehlkopfheilkunde, 128:272, 1931.

Hamburger: Beitrag zur Oberflaechen-Anaesthese in der Rhino-Oto-Laryngologie. Zeitschr. f. Laryngologie, Rhinologie, Otologie und ihre Grenzgebiete, 23: No. 1-2, 1932.

Hopstein: Erfahrungen mit dem Lokalanaesthetikum Larocaine in der Oto-Rhino-Laryngologie. Zeitschr. f. Laryngologie, Rhinologie, Otologie, 21:36, 1931.

Jent: Klinische Erfahrungen mit dem Lokalanaesthetikum Larocaine. Schweiz. med. Wochenschr., No. 14,339, 1933.

Petermann: Erfahrungen mit dem Lokalanaesthetikum Larocaine. Therapie der Gegenwart, 73:526, 1932.

Ridpath: Diagnosis and Treatment of Some Laryngeal Conditions. Penn. Med. Journal, June, 1933.

Schoenlank: Einige Bemerkungen ueber Technik und Dosierung der Larocain-Anwendung in der Otorhinolaryngologie (Zurich). Schweiz. Med. Wochenschr., 63:715, 1933.

Taegen: Erfahrung mit Larocaine. Med. Klinik, 29:362, 1933.

Vock: Ueber Larocain. Zeitschr. f. Laryngologie, Rhinologie, Otologie und ihre Grenzgebiete, 22: No. 4, 1932. Larocaine, ein neues Kokainersatzmittel. Zeitschr. f. Laryngologie, Rhinologie, Otologie, 21:464, 1931.

Fisher and Bledsoe: A Harmless Mucous Membrane Irrigant. J. Am. Med. Assoc., 88:1881, 1927.

Stitt: Rubber Catheter for a Bronchial Tube. Tr. Am. Acad. Opthal. and Otolaryn., 32:369, 1927. Bronchial Lavage for the Disinfection and Immunization of the Bronchial Tree. Cincinnati Jour. of Med., Jan., 1934.

L.

A CLINICAL STUDY OF THE FUNCTIONING OF THE MAXILLARY SINUS MUCOSA.*

EDWARD KING, M. D.,

CINCINNATI.

The function of the sinus mucosa as demonstrated by Hilding, Proetz and others in experimental work on dogs, is well known to all rhinologists. Some time ago Hilding made a clinical observation on a patient who had a large intranasal window in the antrum. He put India ink in the antrum and by means of a nasopharyngoscope watched the flow of the ink past the artificial opening out through the natural orifice.

Recently we published a series of cases studied by means of x-ray with the aid of iodized oil before and after the intranasal antrum operation. In connection with this study a few cases were reported in which an attempt was made to determine whether the drainage from the antrum occurred through the artificial opening or through the natural orifice.

Since then some more cases have been examined and the conclusions are the same as published in the previous publication.

The technic consisted in placing a small quantity, 1 cc. or less, of iodized oil in the antrum in which an artificial opening had been made and observing the flow of the oil out of the antrum by means of a series of x-ray pictures.

In the original communication seven cases were studied. Since then five more cases have been examined. In these last five cases the examination was conducted about three or four weeks after the operation, before the opening had an opportunity to contract. In spite of the large opening the tendency of the oil was to flow out through the natural orifice, although some was found draining by gravity through the artificial opening.

In the original paper two cases were reported in which the oil did not move after a long period of observation (ten hours). Both

*Presented before the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society at Indianapolis, January 23, 1935.

of these cases had been operated upon successfully by the intranasal route, i. e., the symptoms and discharge disappeared and x-rays showed no filling defect in the antrum. It was not understood at that time why the oil did not move. Since then both patients have been under treatment for antrum infection, which recurred almost simultaneously with an acute cold in head. Now we feel that the infection was dormant and the cilia inactive because of the latent infection.

We studied a third case in which the intranasal operation was performed eight years ago. This patient still has a good sized opening, but every time she catches cold it is necessary to wash the antrum frequently. The history showed that following the intranasal operation the antrum did not clear up until strong solutions of silver nitrate had been instilled into the antrum six or seven times. The functional test with iodized oil showed no movement of the oil in spite of the fact that the x-ray picture showed a perfect filling.

The conclusion from this case is that the strong silver nitrate solutions destroyed the cilia and the mucosa no longer functions normally.

We have two other cases in which the intranasal operation was performed successfully and in whom the functional test showed no movement of the oil. These cases are still under observation. They have had no return of symptoms, but we feel that the antrum is still diseased and may flare up at any time.

Hasty, at a meeting in Cincinnati, showed a case in which the filling defect was marked. After the use of iodochorol a few times the swelling disappeared and the antrum mucosa looked normal. This was surprising because it was my opinion as well as that of a number of other laryngologists that when a pronounced filling defect is found in a chronic infection of the antrum the radical operation is indicated.

The treatment of the chronic antrum can be carried out successfully in some cases. We have three cases in which the filling defect was very marked. My own case illustrates that point. We have two others in whom iodochlorol was used exclusively over a period of a few months and the mucosa returned to its normal size. In both of the cases the functional test was made to determine whether the cilia were behaving properly. In one the function was prompt and efficient and in the other the oil did not move. In the latter case the patient returned after some months with a recurrence of the infection and a pronounced filling defect. In my own case the function is normal.

It is our opinion that we can determine whether the antrum mucosa functions properly by instilling a small quantity of iodized oil into the antrum and watching its action by means of a series of x-rays. The benefits of the intranasal operation may be determined by the same method.

1002 Carew Tower.

LI.

A METHOD TO DETERMINE THE PERCENTAGE OF DEAFNESS IN MALINGERERS.

FRED W. DIXON, M. D.,

CLEVELAND.

Many otologic tests have been devised to detect malingerers claiming a hearing loss. By use of these, one is able to arrive at a fairly accurate judgment whether the claim is true or false. In an injured person claiming total deafness in one ear, it can be stated with certainty whether any hearing remains. We are able to testify that hearing does persist in the supposedly deaf ear. None of these tests so devised, however, give any idea of the amount of the deafness. There are cases where due to accident there has been a true hearing loss. These persons have a just cause for complaint and are entitled to compensation. Not every person suffering an injury becomes a malingerer. Most everyone does, however, in his effort to present a good case, overstep himself in claiming too great an injury to his hearing. In the hope of greater reward he is counseled to exaggerate his claim. Some of these people had a hearing defect previous to the accident. In such a person presenting himself several months after the injury it is often impossible to tell whether this hearing loss antedated the injury. If the person to be tested were absolutely truthful in his replies one could easily arrive at an accurate opinion as to how much damage his hearing suffered. Many of the tests devised presuppose a truthful reply to questions. In reality, however, the moment a malingerer detects the sound in his supposedly deaf ear he states that he hears nothing. In most of our present day tests the person examined can easily tell which ear is being tested. If there exists any doubt in his mind in which ear the sound is heard he refuses to answer or to signal until the sound becomes so intense that he will not be mistaken. Such persons are difficult to manage and require much patience. An intelligent malingerer, given a small amount of coaching, can cause considerable confusion in the examiner's mind.

The following test to be performed requires a ruler, the biaural part of the ordinary Boles stethoscope and two tuning forks of the same pitch. Any standard fork with from 128 to 512 double vibra-

tions may be used, but the 256 is the preferable fork. These forks should be approximately the same size, thus causing the same intensity of sound when struck. It is essential to have two tuning forks of the same pitch, as the musically minded malingerer may detect the different tones when presented to his ears.

The principle upon which the test rests is not new. It is that when two tones of the same pitch and intensity are presented to ears of equal acuity that tone which is closest to the ear is heard to the exclusion of the other tone. Also that when the fork is caused to vibrate the tone is heard some distance from the ear and that this tone increases in intensity as the distance is shortened. This is called the range of hearing for that particular fork. In unaffected ears this range should be the same. Where there is a hearing loss this range is shortened.

A person partially deaf in one ear who is not trying to deceive will state that he hears the tuning fork in each ear but that it is less loud in the deaf ear. When testing the two ears simultaneously, since less sound is entering through the deaf ear the hearing range in the good ear is relatively greater. By measuring this increase you are able to determine the loss in the deaf ear.

The procedure is as follows: The patient is blindfolded and the aural end of the Boles stethoscope with short rubber tubes, approximately six inches in length, is inserted into each ear. These ear pieces should fit snugly in the ears to exclude most of the outside noises. A nurse stands behind the patient to be tested holding the free ends of the tubing pointed in opposite directions. A ruler projects beyond the tube which is connected to the normal ear. In this manner the range of hearing is measured in the good ear. This finding is repeated several times and the average range for intensity recorded. Now a second tuning fork of the same intensity is brought out from hiding (it is advisable that he would not know the nature of the test). The tuning forks are held in each hand and struck on the knees so that the volume is the same in each fork. One vibrating fork is held one-half cm. from the end of the tube which is attached to the supposedly deaf ear. The range of hearing is now again obtained by gradually approaching the good ear until the fork is heard. If the ears are normal or equal in acuity the first vibrating fork in the supposedly deaf ear will exclude all sound in the good ear until the tuning fork is brought to within 2 to 3 cm. from the tube. This means that the hearing in each ear is equal, for the average person can detect the second sound in the good ear at this distance. If the range of

hearing is the same in the good ear with or without the vibrating fork in the supposedly deaf ear, then hearing is lost and he is not malingering. Hence the "hearing range" in the normal ear, with the vibrating fork in the supposedly deaf ear, may vary from 1 cm. (normal) to the hearing distance for that fork (complete deafness), depending on the degree of impairment. If with the vibrating fork in the deaf ear, the range of hearing is shortened in the good ear it can be interpreted as a percentage loss in proportion to the shortened distance. For example: If the "hearing range" in the normal ear is 20 cm. but with the vibrating fork in the supposedly deaf ear the hearing range is only 8 cm. it can be stated that he does have normal hearing in one ear with approximately 60 per cent hearing in the other ear.

While this principle is not new, the method has certain advantages in that outside noises are largely excluded, the sound is intensified with the tubing, while the claimant, not being familiar with the stethoscope, is more confused as to the origin of sound. It is impossible for the malingerer to guess accurately and consistently his range of hearing with the opposite ear thus stimulated. One is able to state on the witness stand with a fair degree of accuracy the presence and percentage of hearing loss in a malingerer.

1029 Rose Bldg.

LII.

THE CLINICAL RELATIONSHIP OF INFECTIONS IN THE UPPER RESPIRATORY TRACT TO CERTAIN TYPES OF CHRONIC UVEITIS: A PRELIMINARY REPORT.*

WILLIAM D. GILL, M. D.,

SAN ANTONIO.

Since Rosenow and Billings first pointed the way to a clearer understanding of the relationship between chronic focal infection and the eye, it has become generally agreed that such foci are pre-eminently the cause of certain inflammatory diseases in that organ. The location and duration of the responsible foci are variable, but they have been proven to have a direct causative bearing on such chronic inflammatory reactions by numerous clinical investigators. So far as the eye is concerned the vascular tunic, or the uveal tract, is predominantly the area involved in these so-called elective localizations. Foci in the upper portion of the respiratory tract are of paramount importance in the causation of chronic uveitis, as they are the greatest in frequency of all foci encountered in a search for the underlying cause.

Uveitis is a term used to designate inflammation of the vascular tunic of the eye and is divided clinically into an anterior type in which the iris shows the principal involvement, and a posterior type in which the ciliary body and choroid show the principal changes. Oftimes there is an extension of the pathologic change from one part of the uveal tract to another, because of the close anatomic relationship which exists between them.

Frequently uveitis is considered as a disease which is manifested only by certain classical changes in the anterior segment of the globe, such as deposits on the corneal endothelium, the so-called keratitis punctata, injection of the ciliary blood vessels, inflammatory changes in the iris, and oftentimes coincident alteration in the clarity of the fluid media.

---

*Presented before the meeting of the Mid-Western Section of the American Laryngological, Rhinological and Otological Society at Dallas, Texas, January 28, 1935.

The type of uveitis with which we are concerned is the low grade insidious type involving the vascular structures back in the iris, the so-called posterior type of uveitis, in which the onset is of a relatively painless nature, accompanied by changes in the consistency of the vitreous: this structure becoming abnormally fluid; floating opacities in the vitreous, which vary in degree from spider web strands to veritable sheets of exudate; asthenopic symptoms, photopsiæ and reduction in visual acuity. Quite often there is associated tenderness of the eyeball when it is palpated, but marked pain is usually not encountered. Demonstrable changes in the choroid can oftentimes be made out of the opacity of the media is not too great. Extension to the more anterior or more superficial portions of the globe will often occur coincidentally or consecutively in chronic uveitis.

One hundred and twenty patients with chronic posterior uveitis due to focal infection which form the basis for this paper were subjected to a critical survey with a view to determining the relative frequency of various foci of infection which might have some etiologic bearing on the eye condition. Paranasal sinus infections were surprisingly frequent, occurring in fifty-eight patients, or 48.33 per cent. Of the total number (120) forty-seven patients, or 39.16 per cent, had chronic infection in the ethmoid sinuses. In eleven patients, or 9.16 per cent, the infection was localized in either one or both maxillary antra. There were no patients in which the frontal or sphenoid sinuses alone showed demonstrable infection. The usual type of infection encountered was the low grade inflammatory reaction which is usually designated as latent or concealed infection. In this series of patients there was only one instance of acute sinus inflammation occurring in conjunction with uveal disease, and that was an acute exacerbation in a chronically diseased frontal sinus. Reference to the accompanying table will show the incidence of foci of infection in other parts of the body.

The methods used in diagnosis of paranasal sinus infections were the same in all cases and were based on the usual objective findings, such as the location and character of the nasal discharge, gross appearance of the nasal tissues, cultural and cytologic examination of the discharge, the x-ray findings and transillumination where applicable. In quite a few instances operative verification and histologic examination of the excised soft and bony tissue was made. In the tissues examined the histologic changes were chiefly those of hyperplasis, with round cell infiltration, principally lymphocytic, and with an

especially striking perivascular cuff of leucocytic infiltration which oftentimes extended into the bony tissue.

The average age of the patients was 46 years, the eldest being 86 years and the youngest 13 years. The sex distribution was about equal, there being sixty-six females and fifty-four males represented. The greatest number of cases occurred in the fourth and seventh decades of life.

Evidence of chronic tonsillar infection was present in sixty-one patients, or 58.33 per cent, the only area showing a higher percentage of involvement than the paranasal sinuses.

Some type of dental sepsis was present in thirty-four, or 28.33 per cent, of the patients, which is in conformity with the observations of other writers on this subject.

Constipation is almost universally present in chronic posterior uveitis and it is of interest to note that thirty-one patients, or 25.9 per cent, of the total were suffering from chronic colitis. The colitis was almost invariably of the spastic type and associated with a change in the normal bacterial flora of the intestine. The hemolytic streptococcus was most frequently considered the offender, and it was surprising to find that quite often the same type of pathogenic organisms were present in both the stool culture and the culture obtained from the nasal or sinus discharge. It would appear from this investigation that patients suffering from chronic paranasal sinus disease are especially prone to acquire infection in the lower bowel by swallowing infected secretions over a long period of time and that this infection is most likely to be of the same type encountered in the nasal discharge. There was only one patient in the series in which Bargens bacillus was found in the stool culture. The organism was not present in the nasal discharge. The infection in the colon may often be considered a focus of infection secondary to the primary nasal focus.

In addition to the observations noted above, twenty-four patients were tested intracutaneously with uveal pigment to determine if they exhibited hypersensitiveness to uveal pigment. In all patients tested it was found that some degree of cutaneous hypersensitiveness existed. The usual control injections of 1:20 bovine serum were used and were negative in all cases. The latter cases were not included in the series. The degree of reaction induced by the intracutaneous injection of uveal pigment varied from mild local erythema, with slight induration about the point of injection, to more severe reactions characterized by induration with vesiculation and ulceration.

In two patients constitutional reactions, such as nausea and vomiting, with rise of temperature, followed the skin test. In these tests we have used the so-called normal pigment suspension made from bovine eyes as well as autolysed pigment obtained from the same source, but we have concluded that the normal uveal pigment suspension is most active as a test antigen.

Infection in one or more of the paranasal sinuses which continues for a variable period of time may reach a stage which we designate as chronic, and in which the symptoms referable to the nose may be so mild that they escape the patient's attention entirely or make little impression. In our series of patients the duration of the antecedent sinus infection estimated from the case histories was slightly in excess of two years. It is in such mild sinus infections that elective localization is prone to occur in the vascular tunic of the eye. At the point where this blow is delivered a low grade inflammatory reaction is set up which calls forth a cellular response with infiltration in the area about the choroid vessels with the usual cells characteristic of such reactions. Liberation of various enzymes, proteolytic, saccharolytic and lipolytic, occurs when these cells disintegrate. It is most probable that through their action the protein-like uveal pigment, which is ordinarily nonabsorbable, is acted upon and converted into a soluble substance which is slowly absorbed and acts as an allergin which sensitizes the patient to uveal pigment. Once the patient is sensitized then secondary allergic reactions will be produced wherever a similar type of pigment occurs in the body. Naturally this means that the fellow eye is prone to become affected, and from a practical standpoint this is exactly what occurs, as a very high percentage of patients with chronic posterior uveitis have the disease in both eyes. If uveal pigment is introduced into the skin as a test antigen a reaction characteristic of hypersensitiveness will occur at the point of injection. This would indicate that the remote effect of focal infection which electively localizes in the eye is the production of a special type of allergic reaction, namely, hypersensitiveness to uveal pigment.

Another type of reaction can, according to some observers, be produced through the action of toxins from the focus of infection on tissue of the uveal tract which has become previously sensitized to the organism elaborating it, and would explain the absence of bacteria in such eyes when they are subjected to histologic examination. This theory seems to be borne out in our cases, which have been tested intracutaneously with vaccines made from organisms obtained from sinus discharges, as in the patients investigated in this manner hyper-

sensitiveness to autogenous vaccine was almost universally present. Here it may be relevant to state that heat-killed bacterial suspensions are preferable for skin testing to avoid the confusing reactions which comes from tricresol, as approximately one-fourth of all persons are hypersensitive to tricresol when introduced intracutaneously, even in weak dilutions.

It is interesting to note that pigment of type similar to uveal pigment occurs in the labyrinth of the internal ear, which may partially explain the symptoms of vertigo which is so often complained of in ethmoid disease. Extension of the reactions from the labyrinth to the cochlea may explain the rather frequent association of deafness and uveal disease or offer an explanation for such a condition as Ménière's disease on an allergic basis. Unfortunately our observations concerning the frequency of this condition did not include all the patients in the series, so the exact frequency of its occurrence cannot be stated, as the relationship was not clearly evident early in the series, but from the number of patients in the series that have been investigated from this standpoint we are inclined to believe that demonstrable impairment in hearing is present in well over one-fourth of the patients and that vertigo due to labyrinth irritation is equally frequent. Where labyrinth tests have been employed we have not found any definite diagnostic evidence of disease in this organ other than an occasional instance of hyperirritability, but a more extensive survey on this special point is contemplated in future cases.

The anterior portion of the uveal tract, the iris, apparently does not possess this capacity for producing sensitization to uveal pigment, which is probably accounted for in the fact that there is a structural or chemical difference in the type of pigment present in this structure and the ciliary body and choroid.

It is also of interest to note that fifteen patients, or 12.5 per cent, of the total number had demonstrable cataractous changes in the crystalline lens at the time they came under observation because of their uveitis. It has been noted by practically all ophthalmologists that cataractous changes in the lens supervene with great frequency in patients with chronic posterior uveitis, and indicates most forcibly that cataractous lenticular changes are the terminal event in a chronic low grade inflammation of the vascular tunic of the eyeball which had its inception in a focal infection.

By far the most dangerous feature of uveitis is its relatively painless course, which may and often does influence the patient to

postpone treatment until the condition has reached a stage where irreparable damage has been done. How fortunate then is a patient who has headaches or other annoying symptoms referable to his sinuses which force him to seek relief at an early stage of his illness. It is at such times that a thorough and painstaking examination would reveal ocular pathology when the greatest good could be accomplished by treatment.

So far as treatment of the underlying nasal infection is concerned, it is not considered within the province of this paper to discuss this phase of the subject, as it would be foreign to the title under consideration, but it may be stated that all too frequently the various paranasal foci of infection cannot be cleared up entirely, and in such instances we must be content in minimizing the degree of infection present. The difficulty is greatest where extension into the surrounding bony tissue has given rise to a low grade osteitis of a type which cannot be greatly influenced by operation.

Oftentimes residual infection remains in the paranasal sinuses after they have been operated upon and necessitates careful postoperative treatment to eradicate it or minimize its effect.

This article is intended to correlate certain facts observed in a group of patients suffering from infections in the upper respiratory tract, particularly the paranasal sinuses, with coincident ocular disease, in the hope that such a critical survey would reveal facts which could be put to service in the relief of an unfortunate group of patients which constitute one of the most tragic chapters within the limits of our specialty.

TABLE NO. 1.

Total number of patients with chronic uveitis, 120.
Average age, 46; eldest patient, 86 years; youngest, 13 years.

Patients tested and treated with uveal pigment	24	20 per cent
Patients showing infection in sinuses	58	48.33 per cent
Patients having ethmoid sinusitis	47	39.16 per cent
Other sinuses involved	11	9.16 per cent
Patients showing positive tuberculin reaction	2	1.66 per cent
Patients showing positive Wassermann	7	5.83 per cent
Patients showing tonsillar foci	61	58.33 per cent
Patients showing dental foci	34	28.33 per cent

Occurrence of chronic posterior uveitis by decades:

First	Second	Third	Fourth	Fifth	Sixth	Seventh	Eighth	Ninth
0	7	13	26	20	19	26	7	2
1-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90

TABLE NO. 2.

COMPLICATIONS.

		Per cent			Per cent
Kidney infection	6	5	Retinal detachment	3	2.50
Prostatic infection	3	2.50	Hay fever	2	1.66
Colon infection	31	25.9	Arterial hypertension	5	4.16
Lenticular opacities	15	12.5	Episcleritis	1	0.833
Heterochromia iridis	3	2.50	Acute glaucoma	1	0.833
Ophthalmic migraine	3	2.50	Retinal hemorrhage	2	1.66
Pulmonary tuberculosis	2	1.66	Thrombosis, central retinal vein	1	0.833
Hypothyroidism	2	1.66			
Diabetes	2	1.66			

(Multiple complications not infrequently were observed in the same patient.)

All patients included in this survey were subjected to the usual serologic tests to exclude syphilis, which was found to be present in only seven, or 5.83 per cent, of the total number. In the 120 patients constituting our series there were two patients exhibiting hypersensitiveness to tuberculin, and in those patients there was definite pulmonary tuberculosis, which was not, however, considered positive evidence that their ocular pathology was of a tuberculous nature. This is striking, in view of the fact that the older observers were prone to ascribe such conditions as uveitis to either syphilis or tuberculosis.

There were forty cases in which the uveitis was unilateral and eighty cases in which it was bilateral. The right eye was affected in seventeen instances and the left in twenty-three, in the unilateral cases.

There have been no animal experiments in our work, but there is abundant evidence in the literature of the experimental production of uveitis from toxins, as well as bacteria and foreign protein, to substantiate the hypothesis upon which this investigation is based.

I am deeply grateful to my colleagues who have made this study possible through their whole-hearted co-operation and splendid assistance.

323-325 MEDICAL ARTS BUILDING.

LIII.

A NEW SURGICAL APPROACH TO THE MEDIASTINUM THROUGH THE PYRIFORM SINUS.*

SAM E. ROBERTS, M. D.,†

KANSAS CITY, MO.

I am submitting this brief report at this time because much experimental work is under way but is incomplete.

We have made a careful survey of all the medical literature and we are unable to find any previous reference to this method.

REPORT OF A CASE.

CASE 1.—W. B. M., 14 months old, was admitted to the University of Kansas Hospital, January 8, 1935. The provisional diagnosis was: Postdiphtheritic paralysis, or retropharyngeal abscess.

The child was gravely ill. The outstanding symptoms were dyspnea, cyanosis at times, muffled cry, dehydration and prostration. He was unable to take liquids by mouth except in very small quantities, usually a medicine dropper full at a time.

Present illness: Three weeks before admission to the hospital the child had a severe throat infection. The local physician was called in and his illness was diagnosed as diphtheria with an extensive cervical cellulitis. Twenty-five thousand units of antitoxin were administered.

The following week the child remained quite toxic; was unable to cry and had temperature ranging from 102 to 104 degrees F. On the fourteenth day he was greatly improved and had only one degree of temperature. Also, the cervical cellulitis was subsiding.

Five days later he became acutely ill and again had a temperature of 103 to 105 degrees F.; respiration 34, and pulse 140. His symptoms, as stated above, were more pronounced.

At this time, January 8th, the patient was admitted on the pediatric service of Dr. Frank Neff, at the University of Kansas Hospital. Supportive treatment was given for five days, including hypoder-

*Presented before the sectional meeting of the American College of Surgeons, March 13, 1935.

†Professor of Otolaryngology, University of Kansas.

Fig. 1. Mediastinal abscess Jan. 12, 1935.

Fig. 2. Shows complete evacuation of abscess on fourth postoperative day, Jan. 16, 1935.

moclysis and proctoclysis of saline and glucose. The throat culture showed no diphtheria, but streptococci and staphylococci were found.

The blood picture on January 8th was: W. B. C., 37,600; Polymorphonuclears, 60 per cent; lymphocytes, 31 per cent.

The blood picture on January 11th was: W. B. C., 60,000; polymorphonuclears, 79 per cent; lymphocytes, 16 per cent.

X-ray on January 12, 1935, by Dr. Tice (No. 31975, see illustration No. 1), showed: "No intrinsic lung pathology; mass extending upward from base of heart on right. The shadow is 6.5 cm. in its greatest transverse diameter. The trachea which lies in the midline but in lateral view (illustration No. 3) is seen pushed far forward, forming an arc from below the bifurcation upwards to the pharynx. Mediastinal abscess must be considered as a factor producing the clinical picture."

The child was transferred to my service the evening of January 13th, with the suggestion that a tracheotomy might help. The clinical picture of tracheal obstruction was lacking; the child appeared moribund and any manipulation seemed unwise. However, the next morning he was no worse and a direct laryngoscopy was decided upon.

Fig. 3. Shows anterior arching of trachea and bronchi due to pressure from mediastinal abscess pushing structure forward.

The naso- and oral pharynx were entirely normal; not even any redness or edema. Exposure of the larynx showed the cords to be normal; the left pyriform sinus was normal with the usual concavity. The right pyriform sinus, instead of the normal concavity, showed a bulging convexity; fluctuation of the mass could be demonstrated by palpation with a blunt probe. With a stab knife, through the laryngoscope, the right pyriform sinus was incised to a depth of about 3.5 cm. A gush of thick yellow pus, in an amount estimated at 60 cubic centimeters, filled the child's mouth. The child was suspended by his feet to prevent strangulation. The culture showed streptococci and staphylococci.

Before leaving the operating room the child's breathing was much improved. The foot of his bed was elevated about 60 cm. on returning to his room. Six hours later he was able to take fluids by

Fig. 4. Lipiodol injected in right pyriform sinus of dog. X-ray taken three hours later shows oil in posterior mediastinum.

Fig. 5. Same dog ten days later

mouth, apparently with little discomfort. The second and third postoperative day his intake was normal.

So far, study in the anatomic laboratory shows the distance from the floor of the pyriform sinus to the dome of the posterior mediastinum to be about 6 to 7 cm. in adults.

Under morphin and cocain anesthesia the right pyriform sinus of a dog was injected with 10 cc. of lipiodol, at 3 p. m. Three hours later an x-ray was taken (illustration No. 4), showing the oil well down in the posterior mediastinum, following the normal cervical spinal curve.

Work in the anatomic laboratory shows that the prevertebral cervical fascial planes can easily be separated when approached from above through the pyriform.

Fig. 6. Cadaver.—Catheter in right pyriform sinus entering mediastinum. Forceps in larynx and trachea to show anatomic relationship.

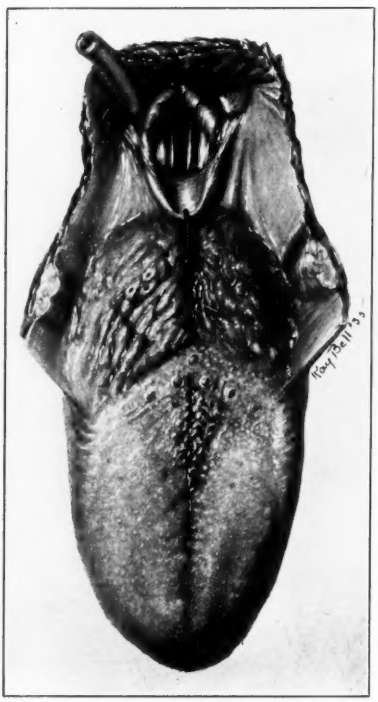


Fig. 7. Cadaver. Catheter in right pyriform sinus. Viewed from above.

After a cross-incision is made in the pyriform a laryngeal dilator was used to separate the planes. Each time a laryngectomy was done on the cadaver and the tract thoroughly inspected. No perforations were found and the dilator easily entered the posterior mediastinum.

Illustration No. 5 was taken ten days later, showing the oil still present, but some absorption had taken place.

It is entirely too early to draw conclusions regarding the clinical application of this approach to the mediastinum. It is reasonable to assume, however, when there is a "pointing" in the pyriform sinus, drainage should be by this route because of its accessibility and simplicity of technic.

Experimental work with valvular drainage tubes, when the mediastinal abscess is not "pointing" in the pyriform sinus, will be carried out.

I want to express my appreciation to Dr. John De Motte, my associate at the university, for his work in investigating the literature and help in carrying on the experimental studies.

Note: Since dictating this article, I have seen Dr. Oscar Batson, professor of anatomy, University of Pennsylvania, and have examined his most excellent anatomic specimens. It is his opinion this surgical approach to the mediastinum is entirely practical from both a clinical and anatomic point of view.

1110 PROFESSIONAL BUILDING.

LIV.

THE IMPORTANCE OF RHINOLOGIC DIAGNOSIS IN ALLERGIC DISEASE.*

HARRY L. BAUM, M. D.,

DENVER.

Allergic disease is fundamental in the individual, whether its symptoms are present in the mucous membranes of the nose and sinuses, the skin, the bronchi, or the intestinal tract. That it is not purely a local condition when occurring in the nose and sinuses seems to require emphasis, and the frequent errors of diagnosis which occur because of the failure of rhinologists to recognize the truth in relation to allergy apparently justify repetition of the statement that allergy is a general disease. It manifests itself in many organs of the body, but probably nowhere so frequently as in the nose and sinuses. Here, strangely, where seen most often, it is very commonly not recognized, and its relation to allergic manifestations elsewhere is almost completely ignored.

I believe that this state of affairs has resulted from several influences, but probably more than any other from the fact that the rhinologist has learned to concentrate his thought and his efforts on the detection and eradication of focal infection. First came the tonsils. Then the sinuses, especially since the observation that sinus abnormalities are associated with frankly diseased states elsewhere.

Too few of us have stopped to classify sinus disease and inquire just what constitutes infection in the sinus cavity or its lining mucous membrane. All deviations from the normal have been called sinusitis, and their acknowledged association with other diseased states is accepted as proof of their etiologic relationship to those states, so profoundly is the profession impressed with the validity of the theory of focal infection. This attitude persists, and from it has developed a train of surgical attacks upon the sinuses, from the ill effects of which neither patients nor profession will recover for many years. But it is time to pause and inquire into the real nature of the pathology in certain of these cases of so-called sinusitis, and their relation to some of the pathologic states associated with them.

---

*Presented before the Dallas Southern Clinical Society, March 21, 1935.

It is only when the individual is fundamentally allergic that the manifestations of allergy will be found in the nose. Therefore, when the patient is known to be allergic or to have a history of allergic manifestations in himself or his antecedents all nasal manifestations should be looked upon with the thought of allergy in the mind of the examiner. On the other hand, when the patient presents himself with a nasal or sinus condition which is recognized as allergic, or suspected of being allergic, the patient and the history of his antecedents should be most carefully studied with the idea that past or present complaints referred to other organs of the body may possibly also be allergic in nature. This attitude on the part of the examiner will in itself save many errors and prevent much loss of time in arriving at a correct diagnosis in the great majority of allergic patients. It will enable the rhinologist to keep in mind the correct conception of allergy as a general disease, and to properly correlate his findings in the nose and sinuses.

Thus, if the patient is referred for examination of the sinuses as possible foci of infection in asthma, and the rhinologist finds that the nose has the appearance of allergy and the sinuses are filled with edematous membrane he will think first that the nasal and sinus pathology may be merely a manifestation of a general disease, the patient's allergic state, and not necessarily the original source of his asthmatic trouble. If the patient comes for examination of the nose and sinuses as possible causes of headache and a history suggestive of allergic disease in the nose is elicited, even though no evidence of that disease is present, it may reasonably be thought that the patient is suffering from allergic migraine, the only hint of which may come from the history or nasal examiation. Or if the patient comes with the hope that a focus of infection may be detected to explain the source of a troublesome skin lesion, and it is found that the nose presents certain aspects of allergy, it is worth consideration that the skin lesion is also allergic and such a suggestion can with propriety be made to the referring physician. Examples such as this might be multiplied indefinitely, but these few should suffice for the purpose. On the other hand, there are, of course, numerous cases coming to the rhinologist primarily for nasal complaints which are allergic. Among these are included hay fever and vasomotor rhinitis, the allergic nature of which is generally admitted. Even in the study of these cases the importance of keeping in mind the general nature of allergic disease is apparent.

A careful analysis of case records in my own practice has indicated that over one-fourth of all patients coming to the rhinologist

with nasal complaints suffer from some form of nasal or sinus allergy. This figure was derived from a study[1] of a series of 700 consecutive rhinologic cases of which 191 proved to be allergic. Of these, over 35 per cent were associated with allergic manifestations elsewhere in the body. It is true that nasal allergies predispose to superimposed infection, and such infections are common in allergic sinuses, averaging over 17 per cent in the series just referred to, but in not one of these cases was it possible to demonstrate that such infections acted as foci. Why it is that obviously infected sinuses are so rarely the source of focal infection is difficult to explain, but it is a clinical observation borne out by the experience of others, notably Anderson[2] of the Mayo Clinic, who reports 400 cases studied without finding evidence of focal infection in a single one.

In order to approach more specifically the practical points of actual diagnostic procedure, I shall now outline the methods used in classifying cases of allergic nasal and sinus disease in my own practice. No effort will be made to differentiate between the various forms of nasal allergy, or to outline methods used in detection of the exciting causes of allergic reactions, as that is outside the province of the paper. However, it should be said that all allergic nasal disease manifests the same general characteristics, which will be detailed.

The patient's presenting complaint may directly suggest an allergic condition, as when he comes with his own diagnosis, such as hay fever, or with definitely allergic symptoms, such as nasal obstruction with sneezing, lacrimation and serous discharge. Especially are these latter symptoms suggestive if they are known to follow exposure to pollens, dog or cat hair, horse dander, feathers or other known allergens, or following the ingestion of certain foods. On the other hand, in the absence of definitely allergic presenting symptoms, we should also be on guard if the patient has a positive allergic personal or family history, and inquiry along these lines should be carefully made. Such a history would include hay fever, vasomotor rhinitis, asthma, eczema, urticaria, angioneurotic edema, frequent unexplained migrainous headaches and repeated bronchial attacks of doubtful origin, especially if the latter are associated with sinus involvement—the so-called broncho-sinus syndrome, most common in children.

On examination, simple inspection of the nose will usually reveal the presence of allergy to the trained observer, even in the absence of history or definite symptoms. The membranes are usually pale and edematous in appearance, and distinctly noninflammatory. There is more or less mucoserous discharge, although the secretion is some-

times purulent because of secondary infection. The greatest edematous change is usually seen in the very chronic cases, and it is then usually accompanied by some degree of fibrous hyperplasia. In all types the edema is most marked in the region of the middle turbinate, but the inferior turbinate is usually also pale and boggy, and even the septal mucosa participates in the allergic change, as may all the nasal membranes. Polypi are frequently present, usually coming from the cavities, but also appearing as excrescences upon the middle turbinates. The inferior turbinates very rarely, if ever, show the presence of true polypi, but it is my opinion that the familiar mulberry hypertrophies so frequently seen on the posterior ends or along the inferior borders of the inferior turbinates are in many cases a late development of nasal allergy. The outstanding point to be stressed in the nasal picture is that of paleness with edema, more marked in the chronic cases but present in all.

The nasal membranes are, of course, often pale in certain nonallergic cases which might be mentioned, notably pulmonary tuberculosis and the anemias, but in these conditions there is no edema, and usually the underlying clinical condition is very apparent and the diagnosis is not in the least difficult. On the other hand, there seem to be certain cases of allergy in which the nasal membranes are not pale but congested. Harold Tobey[3] has called attention to this fact, but in my own experience these cases have been very much in the minority and are usually those suffering from the subsiding stage of a very acute allergic reaction. Whatever the reason, this phenomenon undoubtedly does exist in a few allergic cases. A more common variation from the typical is seen when pale areas appear, mottling the membrane with patches of edema despite the congestion. Also, it should be mentioned that edema of the mucous membranes of the nose and sinuses is occasionally present from causes other than allergy. Mechanical irritation can cause it, pressure of malignant growths may also be a cause, as may chronic infection of various types, notably sinus suppuration and atrophic rhinitis, in which latter disease the formation of ethmoidal and antral polypi is very common, as recently mentioned by Pratt.[4]

In most of the nasal allergies we find at first nothing whatever to suggest sinus involvement, either in the history or in the examination of the nose. The patient has neither pain nor tenderness, and although the mere presence of nasal discharge may suggest sinus trouble, the secretion is not normally purulent unless suppurative sinus disease has supervened. However, my own practice is to radiograph every nasal case studied diagnostically from the point of view

of possible sinus involvement. In doing so, I have found that 89 per cent of all cases of nasal allergy present some evidence of allergic changes in the sinus mucosa. This allergic change is manifested by a thickening of the membrane, which will be found histologically to be always a serous infiltration of the subepithelial connective tissue. This edema gives radiographic findings varying all the way from haziness, indicating slight thickening, to extreme density in those cavities which are completely filled with edematous mucosa. Between these extremes may be found all grades of edema, with discrete rounded polypi showing beautifully at times on the radiographic film.

Ordinarily, the use of contrast media is not needed to demonstrate the changes referred to, but the introduction of radiopaque oils will bring out the thickening very beautifully and convincingly and is a method much to be desired in many cases for a variety of reasons. It should be mentioned in this connection that the displacement method of Proetz cannot be depended upon to fill the sinuses in the presence of allergic reactions, either in the nose or the sinuses themselves. This point has been stressed by Proetz in his papers and in his book[5] on displacement filling, and is, in fact, a point in diagnosis in the use of his method. The reason, of course, is obvious. Edema of the membrane in the region of an ostium operates against the entrance of a diagnostic fluid as it operates against the exit of secretions. Therefore, if it is intended to demonstrate thickening of the mucosa of a sinus by means of a contrast medium and the radiograph, it is usually necessary to introduce the oil by means of a cannula. This is only practical in the antrum and the sphenoid, and as the displacement method will occasionally partially accomplish the desired result in these cases it should first be tried, as it is harmless and simple.

There are, of course, certain cases in which a diagnosis of allergy is not easily made, as, for example, in suppurative sinus disease which has been superimposed upon a low grade chronic allergy and has progressed to such a point that it entirely masks the original condition. In such cases the allergic edema of the tissues so reduces their protective power and so interferes with proper drainage that the membranes quickly succumb to invading micro-organisms and suppuration is engrafted upon what was originally a purely allergic state. Here, in addition to the edema, retained secretion is usually present. This is easily demonstrated by the use of the radiograph, if the proper method is used, and is accomplished simply by taking all sinus radiographs with the horizontal ray. I have described elsewhere[6] a simple

apparatus for the use of the horizontal ray, illustrating the positions which I consider most practical, but for the present purpose it is sufficient to state that fluid levels of contained secretion are easily demonstrable by such means if present in infected sinuses. By placing the head in the position desired, and keeping the ray parallel to the level of the secretion, the latter will, of course, by the simplest of physical laws, be always horizontal and a fluid level will be demonstrated if the quantity of secretion present in the sinus is sufficient to collect as a pool in its most dependent portion at the time of radiography. However, the line of demarcation between the fluid and the thickened membrane will not stand out as it does in the case of opaque filling, and the mucosal thickening in cases with accumulated secretion in the sinuses will for that reason not be so easily defined.

It should be added that in nonallergic suppurative sinus disease with retention of secretion some inflammatory thickening of the mucous membrane is also to be expected, so that the mere presence of mucosal swelling is not by any means pathognomonic of allergic edema in such cases. In addition, as has been mentioned, edema of the mucous membranes occurs from a variety of influences without the presence of allergy. These factors add to the difficulty of diagnosis in allergic edemas of the sinuses, but a presumptive diagnosis of allergy can usually be made independent of the radiographic findings, with all of the evidence at hand.

Microscopic examination of secretions is an additional method of great value in arriving at a diagnosis of nasal allergy. This is true even in those cases having retained secretion in the cavities, though less so when the latter has become purulent in character from invasion of infecting micro-organisms. Cytology in antrum diagnosis has been emphasized by Sewall and Hunnicutt,[7] but not as applied to allergic disease. Leicher,[8] as mentioned by Hansel, Hansel[9] himself, and others have called attention to the cytologic changes to be expected in the nasal secretions from patients suffering with allergic disease of the nose and sinuses. Study of secretions taken directly from the sinuses is of interest and obviously valuable, but it is not essential that smears should be obtained directly from the cavity to be studied. The reason is that secretion from the nose will contain the same cellular elements as that from the sinuses, and the method of obtaining it is simpler. However, in aspirating or washing out a sinus it is interesting to secure some of the secretion for cytologic examination and comparison with the secretion from the nose itself.

In pursuing this method of investigation experience will soon enable the examiner to arrive at very satisfactory conclusions regarding the cytology by simple inspection of a smear stained with Wright's or Giemsa's stain. Normally, the secretions contain only a few round cells and an occasional plasma cell or eosinophil. In allergy the eosinophil is the cell of outstanding importance, and the frequency with which it appears will often point the way to a diagnosis. In frankly allergic states eosinophils will frequently be present in large numbers, accompanied by lymphocytes and plasma cells. The added presence of polymorphonuclear neutrophils, even to the extent of replacing the eosinophils almost entirely, may be taken to indicate secondary infection, while in suppurative sinus disease in which allergy is not a factor the pus cell will be almost the only type of cell present.

It is true that eosinophils are found both in the blood and tissue in a variety of other diseases, notably the parasitic infections, and therefore we cannot consider the presence of the eosinophil pathognomonic of allergy. However, its presence in the nasal secretions and tissues may be taken as very significant, for as a matter of practical fact we know that the eosinophil is very frequently found in the nasal and bronchial secretions and membranes when the latter are the seat of allergic reactions, and also in allergic skin lesions. Therefore, if eosinophils are found in considerable numbers, either in the tissues or the secretions, this finding may be safely taken to indicate the presence of an allergic reaction. On the other hand, absence of eosinophils is not especially significant. I have often encountered frankly allergic nasal cases in which no eosinophils were to be found in the secretions. This is especially true in the more chronic types of allergic nasal and sinus disease and does not by any means disprove the diagnosis of allergy. Large numbers of pus cells in the secretions without eosinophils usually indicates a purely suppurative process which is primary. The presence of eosinophils in what otherwise appears to be a suppurative process may be taken as presumptive evidence of the presence of allergy, upon which has been engrafted a suppurative process.

The histologic study of the tissues themselves will indicate in a similar manner the identity of the reaction with which we may be dealing. Finck,[10] Coates and Ersner,[11] Weille,[12], Hansel[13] and others have done excellent work on this subject. Their studies show that the mucous membranes of the nose and sinuses are normally characterized by the presence of large numbers of lymphocytes with plasma cells, but eosinophils are present only in very small numbers, if at all. My own observation[14] emphasized many years ago that edema is

never a normal characteristic of the nasal and paranasal mucosa, so that if edema and eosinophilic infiltration are present in the same membrane it may be considered strongly indicative of allergy. As has been said, edema may be found in the nasal membranes in the presence of other pathologic conditions, but not usually accompanied by eosinophils. A section of mucosa from the middle turbinate of a hay fever sufferer, for example, will reveal marked edematous infiltration in the tunicą propria in addition to large numbers of eosinophils with lymphocytes and plasma cells.

The membranes in chronic allergic conditions will also present the characteristic edema, but with an accompanying fibrosis, and with a similar preponderance of eosinophils, accompanied by lymphocytes and plasma cells. Very little fibrous hypertrophy is present in the acute allergic manifestations, but in the edemas of chronic allergy we have usually a marked increase in fibrous tissue with epithelial hyperplasia, and sometimes with metaplasia from the normal columnar to stratified squamous. Dilated mucoserous glands are a commonplace, and occasionally one of these forms a small submucous abscess from retention of secretion coupled with the invasion of pyogenic microorganisms. Marked edema with fibrous hyperplasia, or with serous accumulation in the subepithelial stroma, results in the formation of polypi, the polypi themselves presenting the allergic characteristics just described.

The facts assembled in this paper are in the main not new or the result of original discoveries of my own, but they seem to me to form a sufficient basis for the theories advanced. It has been my purpose to so emphasize such facts that the true relation of certain forms of nasal and sinus pathology to associated remote conditions may be more clearly understood, for it is only by such means that we can hope to accomplish the more accurate evaluation of allergic nasal and sinus disease which is so much to be desired in the practice of rhinology.

CONCLUSIONS.

1. Allergic manifestations in the nose and sinuses can be routinely diagnosed by means of history, physical examination, radiography, microscopic examination of secretions and biopsy.

2. Proper correlation of nasal findings with other evidences of allergy which may be present is often essential for a correct appreciation of the problem which presents itself.

3. Allergic nasal and sinus disease is a local manifestation of a general disease, and should be treated primarily as such, surgery being

resorted to only in the presence of obstructive lesions or secondary suppuration, and never solely for the cure of the allergy, or for the removal of focal infection.

510 Republic Bldg.

REFERENCES.

1. Baum, Harry L.: The Incidence of Allergy to Rhinologic Practice. Arch. of Otolaryng., 20:804, (December) 1934.

2. Anderson, C. M.: Suppuration in Paranasal Sinuses as Factor in Focal Infection; Review of Four Hundred Cases. J. A. M. A., 94:1889, (June 14) 1930.

3. Tobey, H. G.: Vasomotor Disturbances of the Nasal Mucous Membranes. Trans. Am. Laryngol., Rhinol. and Otol. Soc., 21:253, 1925.

4. Pratt, John A.: Atrophic Rhinitis. Trans. of the Am. Academy of Ophthalmology and Otolaryngology, 1931.

5. Proetz, Arthur W.: Displacement Method of Sinus Diagnosis and Treatment. Annals Publishing Co., St. Louis, 1931.

6. Baum, Harry L.: A New Apparatus for Roentgenography of the Sinuses. Arch. of Otolaryn., 11:90, (January) 1930.

7. Sewall, E. G., and Hunnicutt, L.: Cytological Examination of the Antrum. Trans. Am. Laryngol., Rhinol. and Otol. Soc., 35:230, 1929.

8. Leicher: Uber Allergische Rhinitis (Ausschliesslich des Heuschnupfens). Zeitschr. f. Hals, Nasen und Ohren., 20:238-250, 1928.

9. Hansel, F. K.: Allergy and Its Relation to the Inflammatory Diseases of the Paranasal Sinuses. Trans. Am. Laryngol., Rhinol. and Otol. Soc., 403, 1930.

10. Finck, H. P.: Tissue Changes in the Nasal Mucosa. Trans. Am. Laryngol., Rhinol. and Otol. Soc., 33:63, 1927.

11. Coates, G. M., and Ersner, M. S.: Occurrence of Eosinophils in the Mucous Membrane of the Maxillary Sinus in Asthmatic Patients. Trans. Section on Laryngol., Otol. and Rhinol. A. M. A., 97, 1929.

12. Weille, F. L.: The Pathology of Allergic Tissue as Seen in the Nose and Accessory Sinuses. Trans. Am. Laryngol. Soc., 225, 1930.

13. Hansel, F. K.: Clinical and Histopathological Studies of the Nose and Sinuses in Allergy. Jour. of Allergy, 1:43-52, 1929.

14. Baum, Harry L. The Histopathology and Histogenesis of Benign Growths of the Nose and Accessory Cavities. Annals of Otology, Rhinology and Laryngology, June, 1922.

LV.

SURGICAL EMPHYSEMA OF THE HEAD, NECK AND CHEST COMPLICATING TONSILLECTOMY, AND A REVIEW OF THE LITERATURE.*

CHARLES L. RUBENSTEIN, M. D.,

SAN FRANCISCO.

Surgical emphysema is considered a rare complication in tonsillectomy. There are not many cases on record in available literature, but those cited present interesting phenomena. The oldest case on record of surgical emphysema dates back as far as 1908.

Parrish reported a case of emphysema immediately following a tonsillectomy which extended rapidly, involving the right eyelid, cheek, face and neck and reaching down as far as the last rib.

Richardson reported a mild case of emphysema which occurred in a male adult one hour after tonsillectomy which involved only the right side of the neck and cleared up in forty-eight hours.

Steward has on record a case of blood extravasation into the subcutaneous tissue of the neck, cheeks and sternum, twenty-four hours after the removal of the faucial tonsils, in a boy seven years of age, which resulted in death.

Woodware, in the British Medical Journal in 1908, reported a case of surgical subcutaneous emphysema of the head and neck following trauma, due to the breaking of a pipe stem and the perforation by a piece just above the anterior pillars of the faucies on one side.

Richards published two cases of surgical emphysema which occurred in the Massachusetts General Hospital. One was that of a young girl, and the other of a young boy. In both cases the complication appeared the morning following the tonsillectomy.

Rosenheim reports a case of emphysema developing the day after tonsillectomy in a patient afflicted with epilepsy.

Martin has on record a case of surgical emphysema of the left orbit and cheek an hour after the patient had blown her nose.

*Read before the Sacramento County Medical Society at Sacramento, Cal., May 24, 1934.

Stera reported a case of a young boy who developed subcutaneous emphysema after tonsillectomy which lasted eleven days.

Von Hofe reported three cases of emphysema which occurred in children, and in all three cases the disturbance was on the right side.

Ehrlich reported a very interesting case in which, after intratracheal anesthesia for a kidney operation, the retroperitoneal tissue was found infiltrated with air which later appeared suddenly in the face and neck.

The purpose of this paper is to place on record a case which appears to the author to be an instructive one.

REPORT OF CASE.

CASE 1.—P. R., laborer, 57 years of age, an arrested case of tuberculosis, suffered an exacerbation following an attack of influenza. After four years he had improved once more and had gained forty pounds. Although there was fullness in the throat and constant expectoration, sputum tests were negative and the patient was considered as suffering from bronchiectasis rather than tuberculosis. On consultation with the attending physicians, it was decided to remove his tonsils, which were large and chronically inflamed.

His bleeding time was normal. Sedimentation test, 15.5 in one hour. Costa, positive. Cloudiness appeared at the fifth minute. On May 4, 1934, under novocain-adrenalin infiltration anesthesia tonsillectomy was performed by the dissection method with the patient seated. There was very little bleeding, the capsular surface appearing smooth and intact. During the operation the patient, despite a dose of morphin and atropin given before the operation, and the admonition to control his cough as much as possible, had coughing spells which complicated the operative procedures greatly. The tonsillectomy finished and a careful inspection of the wound proving satisfactory, the patient was returned to his ward and cautioned again to refrain from coughing as much as possible.

A half hour later I was called to his bedside to find him in great discomfort and anxiety because of a puffiness of the entire face and neck, which was more marked on the right side. Upon palpation there was a feeling of crepitation which was audible and felt distinctly by the patient with each movement of his head in either direction. It was diagnosed as emphysema complicating tonsillectomy. The throat examination revealed nothing abnormal, i. e., no bleeding or oozing. X-ray of sinuses was negative. X-ray of the chest revealed no mediastinal emphysema but showed the presence of a large amount of air in the region of the neck and above it. Temperature 99. Pulse 100—rapid, thready. Sedimentation test 21.5. Costa positive. Cloudiness appeared on the third minute. The chest condition was negative.

Information gathered by the nurse revealed the fact that the patient had coughed continually after being returned to the ward, but that there had been no expectoration. In the evening of the same day crepitation could be distinctly elicited posteriorly and anteriorly as far down as the second rib. The patient recovered within six days.

It seems evident that in this case the entrance of air was through the tonsillar beds. Cough during the operation could have brought about the injury to the inner fascial covering of the superior constrictor, although the capsular surface appeared to be intact. The patient suffered also from impaired nasal breathing due to deviation of the nasal septum to the right. Naturally the violent expiratory effort of his coughing, accompanied by impaired nasal breathing with his mouth tightly shut, was sufficient to force the air to escape into the direction of least resistance, namely, through the muscle fibers of the empty tonsil beds. From here it diffused rapidly through the areolar structures with which the patient was well endowed.

How air reaches the subcutaneous tissue is still a moot question. Richards presents the possibility of air entering either by way of perforation or through straining and coughing, under the visceral pleura over the surface of the lung, reaching the hilus. From here it is reflected up along the great vessels into the mediastinum and then along the fascial planes into the subcutaneous tissue of the neck. Air may also enter the parotid gland, being forced into it through Wharton's duct. It is then diffused into the surrounding tissue.

Some writers contend that the air reaches the subcutaneous tissue through the nose and lacrimal passages. Keen believes that violent expiratory efforts with obstruction lead to distention of the lungs and bursting of one or more alveoli. Air escapes into the interstitial tissues of the lungs, causing interstitial emphysema. With further expiratory efforts more air is pressed through the ruptured alveoli. The emphysema travels along the main bronchi to the hilus of the lungs. From there, he maintains, it reaches the mediastinum and then finds an outlet upward into the subcutaneous tissue at the root of the neck.

Gardner and Jones denote two ways in which air may achieve entrance into the subcutaneous tissue of the lungs:

1. The intrapleural route: The air may pass through the visceral and parietal pleura, but adhesions must exist between the pleura at this point.

2. The extrapleural route: In which case the air sacs on the periphery of the lungs rupture and the air follows the line of least resistance, travels beneath the visceral pleura to the hilus of the lungs, following the reflection of the pleura on the great vessels, enters the mediastinum and then follows the fascial planes into the tissue.

Meyer and Lucke, in reporting cases of subcutaneous emphysema complicating influenza and pneumonia, attribute the mode of origin

either to chemical changes in the blood, which permit the air to escape into the tissues, or to rupture the air vesicles at the apex of the lung or through the extrapleural route.

Volpeau's theory of emphysema in hematomas is that gases are produced by the action of acids, such as lactic acid, on the contained blood. He produced emphysema experimentally by injecting lactic acid into hematomas in animals.

These theories approach the solution to some extent; at least they explain how the air reaches the subcutaneous tissue. However, each case must be considered individually.

I wish to thank Dr. Thoren, the Medical Director of the Weimar Joint Sanatorium and the medical staff for the co-operation that made this report possible.

391 SUTTER STREET.

BIBLIOGRAPHY.

Von Hofe, F. H.: Emphysema of Head and Neck Complicating Tonsillectomy. J. A. M. A., 95:934-935, Sept. 24, 1930.

Rosenheim, S.: Subcutaneous Emphysema of the Neck and Chest Following Tonsillectomy in an Epileptic: Recovery. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 28:337-341, May, 1922.

Parrish, B. D.: A Case of Subcutaneous Surgical Emphysema: An Unusual Complication Following Removal of Faucial Tonsils. Laryngoscope, 20:1046, 1910.

Richardson, C. W.: Tonsillectomy with Consideration of Its Complications. Wash. Med. Ann., 9:85-98, 1912.

Keen, J. A.: Medical and Surgical Complications of Tonsillectomy in Childhood. J. Laryngol. and Otol., 47:1-34, Jan., 1932.

Richards, L.: Subcutaneous Emphysema Complicating Tonsillectomy. Boston M. and S. J., 189:203-203, Aug., 1923.

Ehrlich, S. D.: A Case of Retroperitoneal and Extensive Subcutaneous Emphysema Following Intratracheal Anesthesia with Recovery. Medical Record, 86:926, Nov., 1914.

Alexander, J. H.: Complications Following the Submucous Operation Upon the Nasal Septum. N. J. M. A., 16:765-812, 1911.

Martin, G. E.: Case of Surgical Emphysema of Left Orbit and Cheek. J. Laryngol. and Otol., 43:340, May, 1928.

Dickson, E. A. D.: Emphysema in Left Orbit of Nasal Origin. J. Laryngol. and Otol., 43:341, May, 1928.

Gardner, W. E., and Jones, J. G.: Traumatic Subcutaneous Emphysema. J. A. M. A., 76:503, Feb. 19, 1921.

Clinical Notes.

LVI.

SO-CALLED ANEURYSM OF THE ASCENDING PHARYNGEAL ARTERY: REPORT OF ONE CASE WHICH DEVELOPED UNDER OBSERVATION.*

PAUL B. MACCREADY, M. D.,

NEW HAVEN.

Visible pulsation of a large vessel on the posterolateral wall of the pharynx is not an uncommon occurrence. Various opinions have been expressed as to which vessel produces the pulsation—an ascending pharyngeal, an ascending palatine, an aberrant vertebral or a tortuous internal carotid. The condition causes no symptoms and is usually found by chance. With advancing years and the inclination of arteries to tortuosity, a pulsating swelling occasionally results and is mistakenly called an aneurysm. In view of this misconception and in view of the few cases reported in this country it is felt this case which developed under observation is of sufficient interest to record.

REPORT OF CASE.

History.—J. M. H., a white woman, aged 67, complained of deafness and catarrh. For the past three weeks she had had ringing in both ears which bothered her more than the impairment of hearing. Neither of these complaints was a new complaint but had occurred intermittently over a period of several years. Tinnitus had begun nine months previously but had been greatly aggravated during the three weeks prior to her examination. At no time had she ever had any attacks of dizziness.

The family history was essentially negative. The patient had been married for forty years and had had four children, all of whom died of known causes after reaching adult life. She had never had any miscarriages. She had had no serious illnesses, but she had suffered some in recent years with rheumatic symptoms—never enough to cause real disability.

*From the Department of Surgery, Yale University, School of Medicine.

Physical Examination.—The patient was a medium sized, rather thin female, showing the average musculature of an old person. Both eyes showed cataracts. There was no nystagmus or exophthalmos. The ears were normal in appearance but hearing tests showed a mild catarrhal or middle ear deafness, equal on the two sides. The nose showed a moderate hypertrophic rhinitis involving principally the inferior turbinates. The sinuses transilluminated clearly. The patient's teeth were in fair condition. There were several pulpless teeth but no pronounced gingivitis. The nasopharynx, as seen in the mirror, showed no adenoid tissue but a small amount of generalized mucoid discharge. The pharynx showed tonsils which were small and atrophic. The tongue protruded in the midline and was without tremor. Behind the posterior pillar of the right tonsil on the posterolateral wall of the pharynx there was a mass about 1 cm. in diameter. It was noticed that this mass pulsated synchronous with the heart beat. It was possible to trace out from the appearance of this mass the general course of a blood vessel, and it was my distinct impression at the time that the tumor mass was formed by the kink of some aberrant blood vessel.

The patient was referred to a consulting physician for a general examination. He reported the following findings: There was slight thyroid enlargement. The heart showed a moderate enlargement; the left border of dullness was 10.5 cm. from the midline. The second pulmonic sound was accentuated. There were no murmurs. The rhythm was regular. The blood pressure was 200/110. The lungs showed no abnormalities and likewise the abdomen was essentially normal. A moderate degree of varicose veins were to be noted over the legs, and the joints of the fingers showed some evidence of a chronic arthritis. The hemoglobin was 70 per cent. The urine was normal and the Wassermann reaction was negative. It was the impression of this physician that the hypertension was a definite factor in the production of the tinnitus. It should be pointed out that despite a very careful examination this physician failed to notice the pulsation in the pharynx until after his attention was called to it. This is mentioned to bring out the fact that the pulsation was not at all prominent.

Treatment and Course.—On many occasions during the next twelve months the patient was treated for the deafness and catarrh. The postnasal catarrh subsided very materially during this period. At no time was the patient conscious of any pulsation in the throat and she had no symptoms referable to it. There was no change in the size of the swelling during this time. However, during the fol-

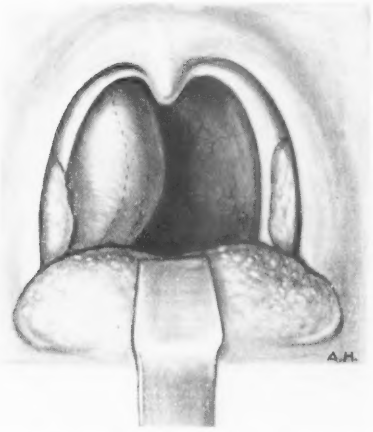


Fig. 1. Large, pulsating swelling on the posterolateral pharyngeal wall of the pharynx as seen before excision. The dotted outline indicates the size and course of the vessel which produced the swelling.

lowing six months' period the swelling in the pharynx seemed to gradually increase in size. In view of this the patient was kept under close observation and the increase in size of the swelling noted from time to time. During the final month before operation—that is, the eighteenth month after the initial examination—the swelling increased so rapidly as to practically double its size in one month's time. It formed a mass the size of an English walnut, reaching to the midline of the pharynx. (See Fig. 1.) There were no symptoms except the sensation of fullness in the throat, as if something were there which should not be there. Despite this absence of any symptoms, the sudden increase in size of the swelling made it seem much more like an aneurysm, and it appeared to be in imminent danger of rupture. In view of this an operation was decided upon, and the patient was referred back to the same physician for a second general examination.

The second examination, eighteen months after the first, showed the following changes: The heart still showed a moderate enlargement, and the blood pressure was 186/106. The lungs showed a moderate amount of emphysema. Otherwise the examination was the same as on the previous visit, except that the swelling in the pharynx, which now reached to the midline, was very evident to the physician.

Laboratory Observations.—The urine showed a very faint trace of albumen with occasional pus cells. Phthalein showed 30 per cent for

the first hour and 25 per cent for the second hour. Nonprotein nitrogen was 21 milligrams. The blood picture showed red blood cells 4,200,000 with 90 per cent hemoglobin, and the white blood cells 5,500.

Operation.—It had not been possible during any of the previous observations to determine exactly which vessel was involved, but having seen the condition develop from what appeared to be a vessel which could be outlined, it was felt possible to ligate this through the pharynx. Despite the numerous observations of Kelly, it was not felt that we were dealing with the internal carotid.

The patient was operated upon under gas-ether anesthesia with exactly the same methods used for a dissection tonsillectomy, such as described by Crowe. A Sewall-Davis mouth gag was inserted after the patient had been put in the modified Rose position. This mouth gag not only holds the mouth open but holds the tongue up out of the way, giving complete exposure of the pharynx and at the same time it carries the ether tube down into the pharynx. An incision was made in the posterior pharyngeal wall below the level of the tonsils and on the proximal side of the swelling. The vessel was doubly ligated with No. 2 chromic catgut. As soon as this vessel was ligated the pulsation of the swelling stopped. The incision was then continued upward over the surface of the swelling. On opening this, instead of finding an aneurysmal sac or a fusiform aneurysm there was merely a continuation of the artery which had been ligated. At no point was this artery increased in size, though it was very markedly tortuous. A second ligature was applied distal to the swelling and the artery between the distal and proximal ligatures was excised. On removal it measured 1¼ inches long. No gross abnormalities could be found in the artery. Unfortunately the specimen was lost in transit to the laboratory so that no microscopic report could be made. The incision was closed with interrupted catgut sutures and the mouth gag was then removed.

The patient made an uneventful recovery from the operation. She was examined on several occasions during the next two or three days, and no evidence of any changes whatsoever dependent upon changes in the blood supply could be found. She was discharged from the hospital on the fourth day.

Postoperative Observation.—The patient has been examined at least once a year for the past six years. She has had no symptoms whatsoever referable to the throat. She does have a persistence of her postnasal catarrh and deafness. On her last visit she stated that she

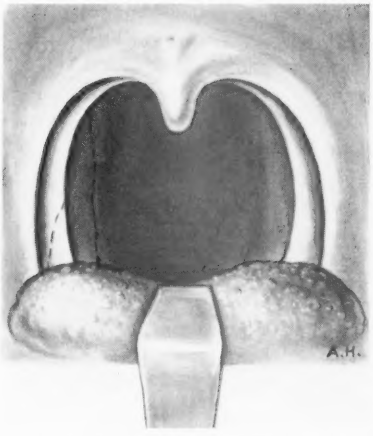


Fig. 2. Pulsation without swelling on the posterolateral wall of the pharynx in a girl aged 21 years. Dotted outline indicates area of pulsation. Pulsation has shown no change during nine years' observation.

felt she had been failing a lot physically. She had lost weight for the last six weeks and she felt she was having some trouble getting her breath due to phlegm in the throat—in reality an aggravation of her heart condition.

COMMENT.

The operation performed does not follow the generally accepted surgical principles. Surgically the carotids should have been exposed from an external incision, and it should have been determined by temporary occlusion whether to ligate the internal carotid or the external carotid. My only explanation is that the success of the operation justified the method.

It is not possible to state even now as to which vessel was ligated. There is no clinical way to determine this. From its size it most obviously was not the internal carotid. It is more than likely, in a patient of this age, that ligation of the internal carotid would have produced cerebral disturbances, though this is merely an assumption. Another fact is that ligation of the vessel proximal to the swelling caused complete collapse of the vessel. This would hardly have occurred if it were the internal carotid. The ascending pharyngeal artery anastomoses with the ascending cervical artery by a small branch, but it most closely approaches a terminal artery of any in that region. Besides the ascending pharyngeal artery normally occu-

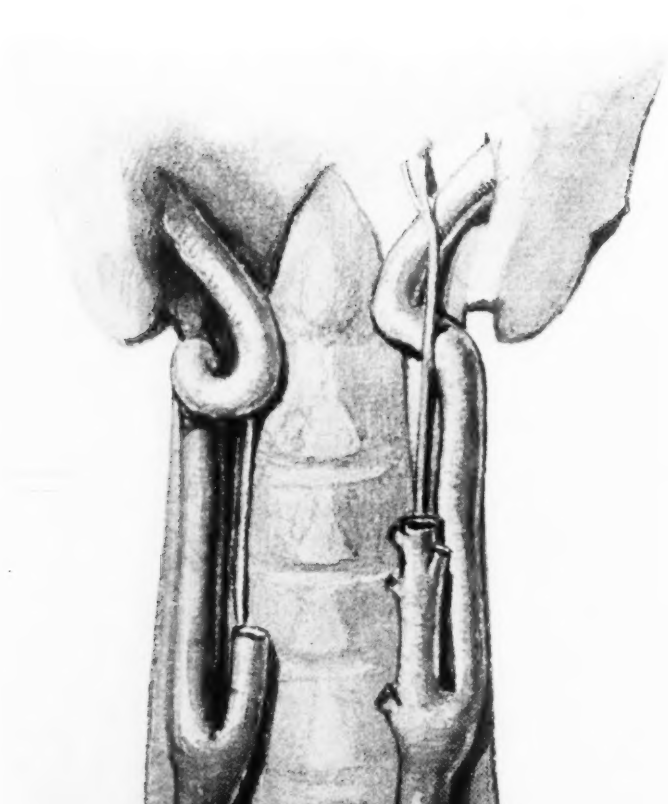


Fig. 3. Tortuosity of the internal carotids which causes the pulsation seen in the pharynx in many cases. (From Kelly—Journ. Laryn.)

pies the position of the vessel under discussion. Brunn and Brown,[1] in 1930, reported what they considered to be an aneurysm of the ascending palatine artery. They were under the impression that no other cases had been reported. From their description one would judge that they were not dealing with an aneurysm but with a tortuous pulsating vessel. While they never determined the exact vessel, at least they abolished the pulsation in the pharynx by ligating the external carotid.

On opening the swelling and dissecting out the vessel there was no aneurysmal sac, not even a fusiform dilatation. A markedly tortuous vessel fairly uniform in size was found to be the cause of the swelling.

This finding of tortuosity agrees with the findings of Kelly,[2] who reviewed the literature up to 1925 and found 150 cases of a pulsating vessel in the pharynx. It differs from his conclusions in several respects. First, after numerous anatomic dissections, he shows that the internal carotid may be much nearer the midline than normally pictured. After explaining this on the basis of an embryonic bend, he states (1) that tortuosities of the internal carotid are more common in the female than in the male; (2) that they are met with mostly in the young and aged; and (3) that they are situated on the right oftener than on the left but may be bilateral. (See Fig. 2.) Secondly, slight tortuosity may remain unchanged for years and then at a later age the artery may incline toward tortuosity, but high blood pressure has not been a prominent factor in most of the reported cases, although it was present in this case. He does not mention the fact noted by anatomists that with the coming on of old age there is a shortening of the vertebral column from atrophy of the intervertebral discs and a tendency to curve forward. This allows the internal carotid artery to curve forward, which would explain its tortuosity in some cases. (Fig. 3.) Thirdly, Kelly could not find a single case in which rupture ever occurred or was imminent. In the present case, which developed under observation, the swelling doubled in size in the last month before operation—reaching to the midline of the pharynx. It was felt that rupture might occur at any time.

This condition under description must not be confused with true aneurysm of the internal carotid artery which projects into the pharynx. The latter may not necessarily be a pulsating tumor and it practically always displaces the tonsil inward. The present condition is a pulsating vessel in the pharynx which causes a bulging or swell-

ing posterior to the posterior tonsillar pillar, especially when the surrounding tissues are thin and atrophied, and which is due to the development of tortuosity of the vessel and not to aneurysmal dilatation.

442 TEMPLE STREET.

REFERENCES.

1. Brunn, H., and Brown, A. L.: Aneurysm of the Ascending Palatine Artery. J A. M. A., 94:29, (January 4) 1930.

2. Kelly, A. B.: Tortuosity of the Internal Carotid in Relation to the Pharynx (Biblio). Jour. Laryn., 40:15, 1925.

LVII.

AUTOPLASTIC NERVE GRAFTS IN FACIAL PALSY.*

JOHN H. FOSTER, M. D.,

HOUSTON.

The objects of this paper are two: First, to pay a humble tribute to the work of Sir Charles Ballance and Dr. Arthur B. Duel in establishing experimentally and practically that the restoration of continuity of the facial nerve is the rational and only thoroughly satisfactory means of dealing with facial palsy in all but exceptional cases; second, to call attention to the fact that this work is possible to any otologic surgeon of fair skill who will master the details of the technic of the operation and the after-care of the wound.

The report of the work of Ballance and Duel[1] represents to me one of the greatest contributions to clinical research in modern times. I have read this publication many times, and each time I get something more from it and am impressed anew by the thoroughness of the experimental work, the true scientific spirit displayed and the soundness of the conclusions reached. In the later addresses of Duel before the American Laryngological, Rhinological and Otological Society[2] and the Royal College of Surgeons of England[3] the progress of the practical application of the results of the research is outlined in a series of cases. In a recent personal letter he informs me that he has now operated on eighty-five cases.

Not less noteworthy and praiseworthy in these reports is the thorough and painstaking detail with which is described every step in the operative technic and the after-care of these cases. This is in marked contrast with the practice of many surgeons who describe the beautiful results obtained by their methods but are so vague in the description of operative technic as to encourage the suspicion that they are deliberately concealing something.

I shall not burden you with a review of the work of Ballance and Duel. Owing to the fact, however, that for years so many of us had the fixed idea that anastomosis operations afforded a satisfactory

---

*Presented before the meeting of the Mid-Western Section of the American Laryngological, Rhinological and Otological Society, Dallas, Texas, January 28, 1935.

solution of the problem, I am taking the liberty of quoting the following conclusions from them: "There can be at the present time no question as to the disability attending an anastomosis operation, and as to the imperfections present in many cases that were hailed as good recoveries in the past. In further considering the future of the operation for the relief and cure of facial palsy, we are driven to the conclusion that the anastomosis operations of the past may be looked on as a stage in the evolution of this branch of surgery." Again, "By exposing and dealing directly with the damaged portion of the nerve, the surgeon's efforts will result in a better recovery; no associated movements or paralysis of muscles subserving other regions will mar recovery. We hold strongly that anastomosis operations for the cure of facial palsy should be banished from future surgical practice. Nevertheless, to every law there are exceptions. When the facial nerve in all its long course within the fallopian canal has been destroyed, for example, by necrosis of the petrous or by gunshot wound, so that an intratemporal nerve grafting operation is not possible, there is nothing left to do but to perform the old operation of anastomosis."

I may say, in passing, that here Ballance ignores completely the efforts of plastic surgeons, notably Sheehan of New York and Gillies of London, to use muscle and fascial transplants to correct the deformity and restore a modicum of movement to the face.

Another fact was brought out by the work of these investigators which is of no less importance from a clinical standpoint than the autoplastic nerve graft operation. I refer to the conclusion that when facial palsy develops, either spontaneously or due to operative trauma, if the nerve is exposed and the sheath incised, the necessity of further operative procedures will be obviated and partial recoveries prevented.

REPORT OF CASES.

CASE 1.—V. W., age 22, consulted me June 8, 1933, with severe pain in his right ear. He had been suffering from an infection of his nose and throat for two weeks and the ear had been affected for two days. Upon examination the tympanic membrane was red and bulging. On incision a copious serous discharge escaped. He was seen the following day, when all subjective symptoms were relieved and the ear discharging freely. He did not return until June 29th, three weeks later. At this time he had a very frank case of mastoiditis, as evidenced by a very copious, thick, purulent discharge from the ear, sagging of the posterosuperior canal wall, tenderness over the whole mastoid process, edema and special tenderness over the mastoid tip,

a dull headache and a temperature of 100° F. X-ray examination showed breaking down of the trabeculæ. Operation was advised at once but delayed by the patient for four days.

On July 3, 1933, the mastoid was opened under ether anesthesia. The cortex was thin and a large pneumatic process was found, full of pus and large granulations. The whole tip was broken down and all of the cell walls soft and easily removed with a curette. The process was thoroughly cleaned out to the inner table and the wound closed with a rubber tissue drain.

The patient awoke from the anesthetic promptly and showed no untoward symptoms during the day, but during that night developed a paralysis of the right side of the face. The nurse thought that when she first noticed it the loss of function was only partial, but the next morning it was complete.

Since the palsy was not present until several hours after operation I thought it was due to postoperative edema and would clear up in a short time, as I had seen in cases of late palsy coming on after radical operations.

The healing of the mastoid wound was uneventful. When the facial palsy showed no sign of improvement I suggested reopening the wound, but the patient did not want it done until we were sure it would not clear up spontaneously. He reported back occasionally for observation and when no improvement was noted after three months, operation on the nerve was urged. He consented, but for various reasons delayed it until March 12, 1934, eight months after the onset of the palsy.

Operation: Under ether anesthesia the mastoid was reopened and a radical operation performed. Flaps were cut from the canal and sutured back. The posterior canal wall was gradually lowered until the facial nerve was exposed. About half way between the knee and the stylomastoid foramen a hiatus in the nerve, almost a centimeter in length, was found. The nerve at either end of the gap was enlarged and bulbous. The nerve was well exposed from the knee to just above the stylomastoid foramen, the bulbous ends were trimmed off somewhat and two strands of the internal cutaneous nerve, removed from the forearm, placed in the bony groove so as to fill snugly the hiatus in the facial. The whole exposed nerve was covered with a strip of gold foil and the wound loosely packed with short strips of gauze wet with normal salt solution. The packing was not disturbed for three days, but was kept moist with saline solution. After three

days the dressing was changed daily, great care being exercised not to disturb the gold foil covering the nerve. At the end of two weeks the foil was carefully removed and the nerve found entirely covered with healthy, pink granulations. The patient was dismissed from the hospital and returned to the office for dressings. Healing was uneventful. Epidermitization was complete in eight weeks. The posterior wound was not closed by a secondary operation for the reason that it looked as if it would close spontaneously. It did not, however, and he has a postauricular opening which he has thus far refused to have closed.

Subsequent history: The patient reported back at fairly regular intervals for observation. For nearly five months there was no change, subjectively or objectively. In August he reported that he could distinguish a difference. The face felt as if it wanted to move and he had a tingling sensation in it at times. No change was apparent objectively. From that time, however, each month there was a visible improvement in the tone of his facial muscles. On November 9th there was some voluntary movement of his lower lid and his face was practically straight in repose. He said that people no longer noticed that there was anything abnormal in his appearance and asked him about it as they did almost daily before. He stated that his face felt as if it were moving and that he often went to the mirror to see whether or not it did, but he had been unable to detect any motion. On November 21st he called up in great excitement to say that he could move his face. He came to the office the next day and he could raise the angle of his mouth and close his eye fairly well. This power of movement was not constant. At times he could exercise it fairly well and again scarcely at all. There was no motion in the occipitofrontalis and his face was markedly asymmetrical on smiling.

During the next two months improvement was gradual, but slower than I hoped for. He still cannot elevate his forehead. He can smile symmetrically, but on laughing the face is drawn to the left to some extent.

Whether or not this case will proceed to complete restoration time alone will tell. I shall watch it with interest and be glad to report further at a later date. It is evident that the nerve has grown through the graft to the face. The explanation of failure of the frontalis to move may be the longer distance for the nerve to go, the failure of certain axons of the nerve to grow out, or to fibrosis of the muscle. If no further improvement occurs the patient will be satisfied.

CASE 2.—Theresa M., age five years, had an operation on July 31, 1934, for acute mastoiditis with a subperiosteal abscess on the left side. The wound continued to discharge for three months, and she was admitted to the Houston Eye, Ear, Nose and Throat Hospital on November 11, 1934, under the care of one of my associates, Dr. J. M. Robison, for a secondary operation.

The mastoid wound was reopened and extensive involvement of the bone found extending into the zygoma and into the bone surrounding the semicircular canals. The softened bone extended very deep, and there was found an exposure of the facial nerve in its descending portion. I was not present at the operation, but a few hours afterwards Dr. Robison informed me of the findings and the fact that a complete paralysis of the left side of the face was found as soon as the child awoke from the anesthetic. At my suggestion the child was taken back to the operating room and the wound reopened. The facial nerve was found exposed for a distance of almost a centimeter midway of the descending portion. It was dark blue in color, looking more like a vein than a nerve, and had a boggy appearance. The bone above and below the exposure was removed for some distance and the sheath of the nerve incised with a fine knife. The nerve was carefully covered and the wound loosely packed with gauze saturated with normal sodium chloride solution.

Subsequent history: The nerve quickly became covered with granulations. In five weeks some motion of the facial muscles was evident and from that time the progress of recovery was rapid. Nine weeks after operation no evidence of palsy could be noted.

COMMENTS AND CONCLUSIONS.

1. Cause of the palsy: A careful study of Case 1 convinces me that the only rational explanation of the sequence of events is that at the time of the mastoid operation a large pneumatic mastoid was found; the cellular structure was badly broken down; a cell extended to the fallopian canal, and in scraping it out the sheath of the nerve was injured; following this trauma the nerve swelled and paralysis developed. Due to this swelling, and possibly infection, the portion of the nerve exposed sloughed out. The nerve could not have been severed or paralysis would have been immediate.

In Case 2 the nerve was probably traumatized a little more severely, as paralysis came on at once. The appearance of the nerve gave evidence that it was already affected, as it was blue and unhealthy in appearance.

I am convinced that the prompt interference in this case prevented the development of the same condition that was seen in Case 1. I feel equally sure that the loss of nerve tissue and the necessity for the autoplastic graft operation in Case 1 could have been avoided if we had handled this case as we did Case 2.

It may be argued that the second case might have cleared up promptly if let alone. Of course it is impossible to prove the point, but the nerve was tightly compressed by the bony canal at either end of the exposure, was swollen, blue and unhealthy looking. It would have been but a step to gangrene and a slough. Be that as it may, the procedure adopted was easy of execution and the result most satisfactory. If I am so unfortunate as to have another case of facial palsy following operation I shall certainly advise immediate exploration. The only exception I should be inclined to make is in a case coming on several days after a radical mastoid operation. I have never had an immediate facial palsy in connection with the radical operation, but have had several, partial or complete, coming on from two to seven days after operation. These have all cleared up completely in a short time and this experience naturally makes me optimistic in this particular type of case.

2. Causes of delay in return of function: (a) Delay in operation; eight months elapsed after the onset of the palsy. It is probable that by that time changes had occurred in the muscles. (b) The use of a fresh graft instead of one in which Wallerian degeneration had taken place. It was not until two weeks after I did this operation that I learned of the new technic advocated by Duel, by means of which he finds the time for recovery is cut to from a half to a third of the time required where a fresh graft is used. (c) I believe that if I had removed all of the enlarged ends of the nerves and had slit the sheath beyond it, it is possible that the outgrowth of axons from the central portion and ultimate restoration of function might have been hastened.

1304 WALKER AVE.

BIBLIOGRAPHY.

1. Ballance, Sir Charles, and Duel, Arthur B.: Operative Treatment of Facial Palsy. Arch. of Otolaryng., 15:1-70 (Jan.), 1932.

2. Duel, Arthur B.: Clinical Experience in Surgical Treatment of Facial Palsy by Autoplastic Nerve Graft: Ballance-Duel Method. Arch. of Otolaryng., 16:767 (Dec.), 1932.

3. Duel, Arthur B.: Operative Treatment of Facial Palsy. Brit. M. J., 3857, Dec. 8, 1934.

LVIII.

UNUSUAL CASES OF CICATRICIAL STRICTURE OF THE ESOPHAGUS.*

HAROLD LESLIE KEARNEY, M. D.,

NEW ORLEANS.

The most common cause of cicatricial stricture of the esophagus is the swallowing of chemical escharotics. Commercial lye is usually the offender, but acid burns of the esophagus are occasionally encountered.

It is noteworthy that these burns from swallowing lye or acids are rarely complicated by obstructive laryngeal dyspnea requiring tracheotomy, despite the fact that the victims are frequently of the tender age when laryngeal obstruction is most easily produced. Hill[1] reported such a case which resulted fatally in an infant that swallowed washing powder. Case 1 is a report of a patient of mine that required tracheotomy for laryngeal edema following the ingestion of sulphuric acid.

REPORT OF CASES.

CASE 1.—A. J., a white male child, aged two and one-half years, was admitted to Touro Infirmary January 4, 1933. He had swallowed an unknown quantity of sulphuric acid about seven hours earlier at his home in Picayune, Miss. The acid (automobile battery acid) had been left in a milk bottle on a porch, where the child found it.

On admission to the hospital his temperature was 103, pulse 200 and respiration 42. There was an ashy gray pallor of the skin and marked obstructive laryngeal dyspnea. There were acid burns of the lips, chin, left eyebrow, fingers and the entire oral cavity. The throat was filled with thick mucus. After a low tracheotomy, which I did at midnight under local anesthesia, the pulse dropped to 114 and the child fell asleep.

Glucose and saline were given intraperitoneally. Mouth feeding was inadequate and gastrostomy was done January 6, 1933, by Dr.

*Presented before the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, at Norfolk, Va., January 19, 1935.

J. D. Rives. The tracheotomy tube was removed January 21, 1933. String swallowing was attempted but was unsuccessful, and January 21st retrograde esophagoscopy was done and a 12 French, Jackson steel stemmed filiform bougie was passed upward into the mouth, where a braided silk string was attached and the esophagus threaded by pulling the string down and out the gastrostomy with the bougie. The upper end of the string was then brought out through the nose and the two ends of the string tied together and looped over the ear, thus preparing the patient for the retrograde method of dilatation of esophageal strictures, according to the technic of Tucker.[5] Retrograde bouginage was begun at 12 French and six months later had progressed to 32 French. Peroral esophagoscopy was not done until July 26, 1933, on account of the earlier feeble condition of the patient, and at that time showed a healthy esophageal mucosa with a stricture located 22 cm. from the upper incisor teeth. Retrograde bouginage was discontinued January 5, 1934, when the 40 French bougie passed easily, and the gastrostomy tube was removed. The gastric fistula would not completely heal due to a small prolapse of gastric mucosa and a plastic closure was done by Dr. Rives July 19, 1934. Esophagoscopy done at this time revealed a lumen so large that the location of the stricture was difficult to determine.

Cicatricial stricture of the esophagus is a rare complication of scarlatina. Vinson[7] reports two cases, one of recent origin, in a child of eight years, and the other in a woman of thirty-eight years who had had recurring attacks of dysphagia since scarlet fever thirty-one years prior to his examination. Following is the history of a similar case which came under my observation:

Case 2.—Mrs. L. D. L., a white female, aged twenty-five, had had scarlet fever complicated by abscesses in the neck, at the age of fifteen years. Three months after this illness she began to have difficulty in swallowing, and from that time her food had to be finely divided for her to be able to swallow it. She at times resorted to massage of the neck in the suprasternal region to assist swallowing. Esophagoscopy revealed a 14 French cicatricial stricture of the esophagus about 4 cm. below the cricopharyngeus. Peroral esophagoscopic bouginage was done on four occasions, but was discontinued because of lack of progress and dislike of the patient for the procedure. Gastrostomy was then done by Dr. Faust, June 20, 1928. Retrograde bouginage was begun nineteen days later with the Tucker 14 French bougie and had been carried to 32 French, the largest size of retrograde bougie then manufactured, about six months later. The gastric fistula was allowed to close and bouginage was continued up

to 45 French by allowing the patient to swallow, under supervision, long woven silk bougies. Two years after dilatation was discontinued in this case, a 9 mm. full lumen esophagoscope was passed to the stomach with no resistance whatever at the stricture level.

Cancer may develop at the site of an old cicatricial stricture of the esophagus. In these cases two types of precancerous lesions may be noted—the cicatrix itself and the static esophagitis resulting from esophageal stasis in small lumen strictures. Magnusson[3] has reported a case of carcinoma of the esophagus in a man forty-one years old, in which case the tumor was located in the neighborhood of a cicatrix from lye swallowed at the age of eighteen months. Myerson[4] has reported carcinoma of the esophagus in a man twenty-nine years old, growing at the site of a cicatrix resulting from swallowing lye at the age of three years. The clinical appearance of the lesion in this case did not definitely suggest malignancy but the microscopic diagnosis was epithelioma.

A patient observed by me developed carcinoma of the esophagus at the site of a cicatrix from lye burn forty-one years after swallowing lye. His history follows:

CASE 3.—H. R., a colored male, aged forty-five years, had had much difficulty in swallowing for two weeks and had lost much weight. He swallowed lye at two years of age and for five years after that was limited to semisolid diet; after that he ate everything, but occasionally had some difficulty in swallowing. Skiagraphic examination showed a very much dilated esophagus in its upper third with a constriction approximately at the aortic arch. At esophagoscopy a slightly bleeding, fungating, infiltrated area was found on the anterior wall of the esophagus slightly above the level of the aortic arch. Clinically the lesion was malignant and biopsy showed squamous cell carcinoma.

The esophagoscopic findings in this case confirm Tucker's[5] conclusion that the final diagnosis in "stricture" of the esophagus should be made by roentgen ray study and direct esophagoscopic inspection. Jackson[2] emphasizes the folly of attempting to make a diagnosis of esophageal disease without looking at the esophagus.

Treatment of esophageal stricture should be promptly carried out by someone trained in the management of these cases. The malnutrition of these neglected and sometimes mistreated little unfortunates, who have long been fed on a totally inadequate liquid diet, is pitiable. The following history is an example:

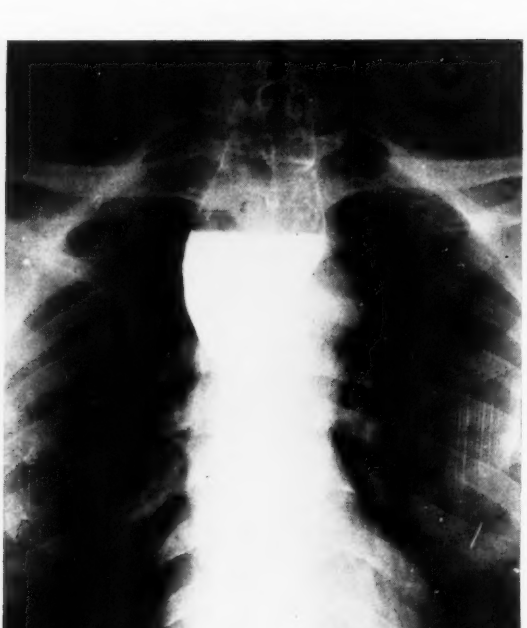


Fig. 1. Case 3. In which carcinoma developed at the site of an esophageal stricture 41 years after the patient swallowed lye.

CASE 4.—M. S., a white female child, aged seven years, developed an esophageal stricture after swallowing lye February 17, 1929. For one year unsuccessful attempts were made by her physician to dilate the stricture by peroral string guided bouginage. She was then taken to a chiropractor, who treated her for two years by rubbing the stomach and the throat. She then again consulted a physician, gastrostomy was done and retrograde dilatation was carried on for two years to 24 French. According to the mother's story, no attempt was made to go beyond that point, as the attendant considered further dilatation too dangerous.

She came under my observation February 16, 1934, a stunted little seven-year-old girl, weighing thirty pounds. Fluoroscopic examination of the esophagus showed a stricture just below the cricopharyngeus. Retrograde bouginage was started at 22 French, and November 5, 1934, had progressed to 40 French. This five-year-old

cicatrix was the most obstinate, unyielding, cicatricial stricture of the esophagus that I have encountered, but the result has been entirely satisfactory and the little patient now eats everything in an ordinary diet and has gained about ten pounds in weight. The gastrostomy tube was removed January 7, 1935, to allow the gastric fistula to close.

SUMMARY.

An account has been presented of the following cases:

1. A child who swallowed sulphuric acid and required tracheotomy for obstructive laryngeal dyspnea, gastrostomy for feeding and retrograde esophageal bouginage for cicatricial stricture of the esophagus.

2. A case of cicatricial stricture of the esophagus resulting from scarlet fever.

3. A case of carcinoma developing at the site of an esophageal stricture forty-one years after the patient swallowed lye.

4. A case of lye stricture of the esophagus of five years' duration presenting unusual features.

204 MEDICAL ARTS BLDG.

BIBLIOGRAPHY.

1. Hill, F. T.: Three Unusual Consecutive Endoscopic Cases. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:94-101, March, 1932.

2. Jackson, Chevalier: Bronchoscopy and Esophagoscopy. Ed. 2, Philadelphia, W. B. Saunders Co., p. 287, 1927.

3. Magnusson, R.: Cancer of the Esophagus After Corrosion with Lye. Acta chir. Scandin., 69:473-478, 1932.

4. Myerson, M. C.: Carcinoma of the Esophagus Engrafted Upon a Lye Stricture of Twenty-six Years' Duration. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 42:1224-1226, December, 1933.

5. Tucker, Gabriel: Cicatricial Stenosis of the Esophagus, with Particular Reference to Treatment by Continuous String, Retrograde Bouginage with the Author's Bougie. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 33:1180-1223, December, 1924.

6. Tucker, Gabriel: Strictures of the Esophagus: Diagnosis and Treatment. Laryngoscope, 41:426-439, June, 1931.

7. Vinson, P.: Stricture of the Esophagus Following Scarlet Fever. Laryngoscope, 33:949-951, December, 1923.

LIX.

CYSTS AND FISTULAE OF THE NECK.

ROLAND D. RUSSELL, M. D.,

CHICAGO.

Early in embryonal development the cephalic extremity of the foregut expands to form the primitive pharynx, in the lateral walls of which shortly thereafter five branchial pouches form, and at the same time externally, opposite the first four branchial pouches, the branchial clefts appear.[1] Craniad to each branchial pouch, except the fifth, is the corresponding branchial bar or arch; in the case of the fifth pouch the corresponding arch is external. The first and second arches, known as the mandibular and thyroid arches, respectively, grow much more rapidly than the inferior arches, so that the latter become hidden in a depression, the precervical sinus, which, as development proceeds, is converted into first a pouch communicating with the surface by the precervical duct, and then, due to obliteration of the duct, into a hollow vesicle, the precervical vesicle, communicating with the second and fourth cleft by means of the narrow branchial ducts. Later the branchial ducts and vesicle disappear, but it may be that part of the thymus is formed from the wall of the vesicle. The wall, or separating membrane, between the clefts and pouches is formed from without in, of ectoderm, mesoderm, and entoderm; the mesodern then disappears but reappears later. Internally the fourth and fifth pouches open into the pharynx by a common aperture, the pharyngobranchial duct. Before the disappearance of the fourth and fifth pouches, diverticula, which later become the solid epithelial bodies, are formed from their dorsolateral walls. These epithelial bodies enter into the formation of the thymic and parathyroid glands. A diverticulum formed from the fifth pouch becomes the ultimobranchial body, which is closely associated with the thyroid gland, but does not take part in its formation in man. The second branchial pouch forms the supratonsillar fossa, and the first pharyngeal pouch the auditory tube and the tympanic cavity. The auricle is formed from the margins of the first cleft, and the external auditory meatus is derived from the first cleft, while the tympanic membrane arises from the first separating membrane. The first arch forms the malleus incus and a portion of the upper and lower jaws, parts of the lips, cheek, chin and the sphenomandibular ligament.[2]

The second arch forms the lesser hyoid cornu, part of the styloid process and the stylohyoid ligament and probably a portion of the stapes. The third arch gives origin to the greater horn and the body of the hyoid bone, while the fourth and fifth arches go into the formation of the larynx.

The second and third branchial arches, together with the furcula, enter into the formation of the tongue. At the location of the foramen cecum a column of cells passes caudad, as the median thyroid anlage, part of the caudal portion of which may remain as the pyramidal lobe of the thyroid gland. The lateral lobes of the thyroid are derived from the caudal extremity of the thyroglossal tract. According to Wenglowski,[2] aberrant thyroid glands may be formed anywhere along the course of the thyroglossal tract. This investigator showed that the lingual duct developed independently of, and at a little later period than, the beginning of the thyroglossal tract, as a sinking in of the oral epithelium from the foramen cecum. The course of the lingual duct is directed anterior to that of the thyroglossal tract, and has more or less branches lined with stratified, ciliated or mixed types of epithelium. As the hyoid bone develops, it pushes forward into the area of the thyroglossal tract and divides it into two portions, an upper and a lower. The upper and lower strands may remain connected, or may be entirely separated by the hyoid body. Both upper and lower strands are usually attached to the periosteum of the hyoid bone, or the strand may even pass through the body of the bone.

The formation of neck cysts and fistulæ may be brought about in a number of ways: epithelial rests may be pinched off from any of the branchial pouches or clefts; a portion of the precervical sinus may remain; the anlagen of the thymus, the parathyroid glands or the ultimobranchial body may leave epithelial rests in the course of their development and migration; cysts may form anywhere along the course of the thyroglossal tract. These latter cysts may be of the nature of thyroid cysts,[4] or of cysts lined with stratified squamous,[5 and 7] ciliated[1] or mixed types of epithelium.[3] In addition to the cysts which arise from embryonal rests, retention cysts of the submaxillary gland may arise. Sebaceous cysts of this region are superficial and although they are not very common, present no diagnostic or therapeutic problem.

Cysts and fistulæ of the neck usually are present anterior to the sternomastoid muscle, and between the lower jaw and the suprasternal notch. Branchial cysts proper frequently communicate with the

supratonsillar fossa,[6] and are often intimately related to the large vessels and nerves of the neck.[7] The thyroglossal cysts, as mentioned above, may form anywhere along the course of the thyroglossal tract,[4] and may lie in the midline or lateral to it. Those above the hyoid bone are said to lie in the midline; however, in case 5, the cyst which, from its attachment to the hyoid bone, must have been a suprahyoid thyroglossal cyst, was singularly placed in the submaxillary triangle. Thyroglossal cysts and fistulæ below the hyoid bone are usually slightly to the right or left of the midline, because they are probably pressed to one side by the prominence of the thyroid cartilage. In the three cases cited below they were placed to the right of the midline.

The various cysts and fistulæ of the neck vary in structure. The epithelial lining may be of the stratified squamous[7] or ciliated columnar variety, or the cyst may be lined in places by stratified squamous and in other places by columnar epithelium or, due to inflammation the lining epithelium may be absent,[4] as in case 1. The epithelium usually lies on a more or less distinct basement membrane with sometimes a muscularis mucosæ.[7] The outer layer is composed of fibrous tissue, usually containing more or less lymphoid tissue, into which mucous and serous glands may penetrate, and the cyst wall may be infiltrated with leucocytes when inflammation exists. The cyst or fistula wall may include striated muscle fibers, which when present are probably derived from the surrounding muscles of the neck. The cyst contents vary in character, depending on the character of the lining epithelium and the presence or absence of infection. The contents may be serous or syrupy, with whitish, yellowish or brownish color. The contents of cysts lined with squamous epithelium may be of a more or less solid whitish or yellowish granular material, due to desquamation of epithelium. Any of the cysts may contain more or less desquamated epithelial cells and leucocytes and considerable cholesterin.

Seelig[8] reconstructed a thyroglossal fistula and found that there were many side branches which themselves divided into secondary branches, and concluded that it was useless to inject corrosive agents into the fistulous tract, as these agents could not possibly reach all the various branches and destroy all the lining epithelium. However, Cutler and Zollinger,[9] Schwers[10] and others report successful obliteration of cysts and fistulæ by injection.

The fistulæ almost always begin as cysts, and are converted into fistulæ by surgical incision, accidental puncture, as in case 3, or spontaneous rupture, as in case 4.

The diagnosis of a cyst or fistula of this region offers, as a rule, no great difficulty, although it may be confused with solid tumors or tuberculous abscesses, in the latter especially in a tuberculous patient, as in case 2. A puncture with withdrawal of fluid for examination may be of help, since the finding of cholesterin crystals or epithelial cells is diagnostic of a cyst, but it should be noted in this connection that the contents of some cysts, as in case 5, are so solid that they cannot pass through the needle. Injection of opaque iodinized oils outlines the cysts in the x-ray film, but the cyst should be resected without delay after this procedure, for the iodinized oil may excite considerable inflammatory reaction. This procedure differentiates between a cyst and a cervical tuberculosis;[11] in the former, the outline is regular, and in the latter irregular. Infrahyoid thyroglossal cysts can be felt to move upward in swallowing, as a rule, because of the attachment to the hyoid bone.

The treatment is generally surgical. For cosmetic reasons the skin incision should be usually transverse; however, in the case of fistulæ a more or less vertical incision may facilitate removal of the fistulous tract. In case of thyroglossal cysts and fistulæ, as pointed out by Sistrunk,[12] it may be necessary to resect a small portion of the body of the hyoid in order to remove the cyst or fistula completely. This procedure is absolutely indicated in those cases of cysts and fistulæ which have both an intra- and suprahyoid extension.

REPORT OF CASES.

CASE 1.—A white railroad yardmaster, aged 36 years, was admitted to the hospital December 17, 1931, complaining of soreness in the right side of the neck of six weeks' duration. One week after the onset of the soreness a tender swelling appeared and gradually grew larger.

General physical examination revealed no findings worthy of record.

The septum was slightly irregular. There was a scant thin mucus discharge in both nares, and x-ray examination showed evidence of a right chronic maxillary sinusitis. The tonsils were large and irregular, the right tonsil being more prominent than the left one. Externally a large mass was seen below the right ear; to palpation the mass felt like a sac of gelatin.

The Wassermann and Kahn were negative. Red blood count was 4,810,000. White blood count was 6,800; polymorphonuclears, 74 per cent; small mononuclears, 22 per cent; large mononuclears, 3 per cent; eosinophiles, 1 per cent, and hemoglobin, 80 per cent.

January 6, 1932, aspiration of the cyst contents revealed a thick dark brown fluid, microscopic examination of which showed numerous pus cells and a few red blood cells. Gram stain showed a few gram positive diplococci; stain for tubercle bacilli showed none. Wright's stain showed chiefly polymorphonuclear cells. A culture showed no growth.

January 18, 1932, the cyst contents were aspirated and iodopin injected. Microscopic examination of the fluid showed cholesterin crystals, a few gram positive cocci and no tubercle bacilli. X-ray examination showed a large sac, containing opaque material, with a fluid level opposite the second and third cervical vertebræ.

A diagnosis of branchial cyst was made and excision advised.

Operation, as well as diagnostic procedures, had been delayed often by exacerbations of an evident inflammatory reaction of the cyst, but January 21, 1932, under local anesthesia, a diagonal incision was made over the cyst, dividing the skin and fascia, the sternomastoid muscle was retracted, and the external portion of the cyst dissected free from the surrounding structures. When about two-thirds of the cyst had been freed, it was ruptured with evacuation of a thick yellow fluid resembling the yolk of an egg. The cyst was traced upward to the region of the right tonsillar fossa. Posteriorly, it was so densely adherent to the deeper structures of the neck that it could not be separated, and the effort at complete resection was desisted from after very troublesome bleeding was encountered. About one-fourth of the cyst wall was not removed by dissection, but an attempt was made to destroy the mucous lining by surgical diathermy. Rubber drains were placed in each end of the wound and the wound closed by subcutaneous catgut and external dermal sutures.

Microscopic examination showed a violently inflamed cyst wall. Practically all the epithelium had sloughed, only one small island of epithelium adjacent to the lumen being seen. There was an exudate on the inner surface of the cyst consisting of pus cells, fibrin and hemorrhage. The wall was markedly infiltrated by fibrin and leucocytes. The blood vessels were engorged and there were numerous hemorrhagic areas. In one place there was an abscess in the cyst wall.

February 15, 1932, the tonsils were removed.

The patient made an uneventful recovery from the latter operation, but the wound of the first operation drained persistently for some time. By March 14, 1932, the wound had completely healed and there was no evidence of recurrence.

Some months later it was learned that the patient had a recurrence for which he was being treated elsewhere. The ultimate outcome is not known.

In this case a corrosive solution might have been more successful than an attempt at resection.

CASE 2.—A Bulgarian chauffeur, aged 35, was first seen July 7, 1930. His family history was negative. He had been in good health until three months previously, when he was confined to bed with pain in the right axilla, painful breathing, weakness, loss of appetite, and productive cough. He soon developed a sore throat, which was diagnosed August 22, 1930, as tuberculous laryngitis, at which time the epiglottis, particularly on the left side, left aryepiglottic fold, left arytenoid and interarytenoid area were markedly swollen and infiltrated. The laryngeal lesion became gradually worse, until the patient left the hospital, some months later, to return to his home in Bulgaria. While in the hospital it was discovered that the patient had a small cystic swelling to the right of the thyroid cartilage which he insisted on having removed.

September 23, 1930, under local novocain anesthesia, a curved transverse incision was made at the level of the upper border of the thyroid cartilage, and afterward a vertical incision was made at right angles to the first incision in order to follow the cyst downward in the pyramidal lobe of the thyroid gland. The cyst fused below with this structure without a sharp line of demarcation. Above it passed anteriorly over the hyoid bone to be attached firmly to its posterior superior aspect. The cyst contained a milky fluid. The incision very promptly healed.

CASE 3.—Aged 32, was admitted to the hospital, December 26, 1931, for treatment of an abscess in the right axilla.

The patient's mother died of tuberculosis at the age of 35, and the paternal grandmother died of tuberculosis.

The patient had had all the diseases of childhood. He had had influenza in 1918. He had a chronic cough without loss of weight. The patient had had a swelling in the right side of the neck for many years. About twelve years ago he had been struck accidentally in the neck with a sharp piece of wood which ruptured the cyst, since which time it had discharged continuously.

Examination showed a well developed, well nourished man who did not appear very ill. There was an abscess in the right axilla. There were a few scattered rales in the lungs. There were no glandular

enlargements. The Wassermann and Kahn tests were negative. To the right of the midline over the thyroid cartilage there was a depressed, umbilicated scar, and above this a small swelling, by pressing on which a milky fluid was evacuated through the scar.

February 6, 1932, under local novocain anesthesia, the scar was freed from the skin and the fistulous tract followed upward behind the hyoid bone to an attachment to the posterior aspect of this structure. The patient left the hospital about one week later with a slightly draining wound.

CASE 4.—A white, rather poorly nourished, senile appearing male, aged 57 years, appearing chronically ill, who came complaining of a discharging fistula in the neck, a chronic sore throat, weakness and fainting.

There was nothing of significance in the family history.

The patient had had measles, mumps and diphtheria in childhood. He had had yellow fever in 1898. Several years ago he had had an ulcer of the rectum with rectal hemorrhages. He had had a herniotomy and an appendectomy in 1922.

The patient first became aware of the cyst when he was sixteen years old, and attributed its occurrence to the wearing of a brass collar button which made his neck sore. A swelling appeared and ruptured spontaneously. It was treated at the time with cessation of the discharge until eight or ten years ago, when the fistula reopened. Since this time there has been constant drainage and he has had frequent sore throat.

Examination revealed a scarred depression about one inch to the right of the midline at the level of the thyroid notch, from which a whitish mucus containing material exuded; on applying pressure over this area a discharge of the same character could be seen with the mirror coming from the foramen cecum. There was another scar, apparently without fistula formation, extending upward a short distance from the sternal notch. The tonsils were large, the crypts contained débris and the pillars were reddened. There were three periapical foci of infection and a gingivitis.

Wassermann and Kahn tests, blood counts and urinalyses gave negative findings, and general physical examination revealed nothing worthy of note here.

Diagnoses of fistula, thyroglossal, complete; and tonsillitis, chronic, were made.

October 25, 1932, a tonsillectomy was performed, and November 3 to 11, dental extractions and treatment of the gums were done.

November 15, 1932, under local anesthesia, a collar incision was made around the external opening of the fistula. A transverse incision was made in a fold of skin in the hyoid, and a third short vertical incision made joining the first two incisions. A probe introduced into the fistula passed to the mid-portion of the hyoid bone. The tract was dissected up to the hyoid bone to the posterior inferior aspect of which it was firmly attached. About 0.75 cm. of the middle portion of the hyoid bone was removed. Above the hyoid bone a distinct cord-like strand could be identified. This was followed up to the foramen cecum and excised. The wound in the tongue muscles was closed with catgut sutures and the fragments of the hyoid brought together by suturing the periosteum. The skin wounds were closed with interrupted sutures.

The postoperative reaction was severe, the patient being unable to swallow for three days, and examination disclosed edema of the epiglottis.

The edema of the epiglottis subsided rapidly and the wound healed readily. The patient has been seen several times since his discharge, the last time being in September, 1934, and at no time could any evidence of a recurrence be elicited.

CASE 5.—A public accountant, aged 32, came complaining of a swelling below the lower jaw on the right side. His family history was negative. He had had measles and chickenpox in childhood and scarlet fever and influenza in the service. He had had several severe colds and tonsillitis, for which a tonsillectomy had been performed.

Nine years previously the patient had had a sore throat, and a lump appeared beneath the right lower jaw. The sore throat disappeared but the lump remained. The size of the lump did not change until about one year ago, when it began gradually to increase in size, without pain but with a sensation of fullness.

Examination revealed a well developed, fairly well nourished white man, who did not appear acutely ill, and whose general physical examination was negative, with the exception of evidence of mild chronic fibrous pleurisy.

There was found an egg sized swelling in the region of the right submaxillary gland, which had the feel of a cyst. By indirect laryngoscopy the epiglottis was found to be pushed slightly to the left.

At operation, July 21, 1931, a direct laryngoscopy was performed with negative findings, probably because the mere passing of the laryngoscope displaced the mass externally.

A transverse incision was made in the fold of skin in the right submaxillary region, the fascia and platysma divided, the submaxillary gland exposed and retracted upward, and the underlying cyst dissected out. The cyst lay directly beneath the submaxillary gland, in the submaxillary triangle.

The cyst extended downward and forward, to be attached firmly to the posterior superior aspect of the body of the hyoid bone. The cyst was well encapsulated. The cyst cavity was filled with a firm semi-solid mass of slightly yellowish granular material resembling cooked rice.

A portion of the dead space was obliterated with catgut sutures and the skin closed with dermal sutures, after inserting a rubber drain in the outer angle of the wound. The incision promptly healed and the patient was discharged July 29, 1931.

Microscopic examination of the contents of the cyst showed it to consist chiefly of desquamated epithelial cells, with cholesterin crystals, resembling in appearance a cholesteatoma.

The patient was seen again August 8, 1934. The scar was to be detected only by very close inspection. There was no evidence of recurrence.

CASE 6.—A white male came complaining of a swelling in the neck. His family and past history were negative, except for occasional rectal pain since 1920, attributed to hemorrhoids.

About two and a half years ago he noticed what he described as blisters under his tongue. At that time there was a small nodule near the midline beneath the tongue, with a soft swelling under the chin. The "blister" had disappeared, but recurred from time to time, and the swelling under the chin remained. Examination revealed a short, well developed and nourished white man, who appeared chronically ill, with negative general physical findings.

The tonsils were large and the pillars reddened. There was a soft fluctuant mass in the mid-hyoid area, which did not bulge into the floor of the mouth on applying pressure, and no mass could be seen or palpated in the floor of the mouth.

At operation, August 18, 1931, a curved transverse incision was made above the hyoid bone and the cyst dissected out. The cyst lay

on the mylohyoid muscles, extending around both sides of the neck, upward beneath the angle of the jaw on the right side, and downward as far as the thyroid cartilage to which it was not attached. The right submaxillary gland was not found. The wall of the cyst was quite thin in places, but in other places it was thicker, being reinforced by muscle fibers. A thick yellowish fluid was present in the cyst cavity. At one place a nodule, which microscopic examination showed to be a lymphatic gland, was attached to the cyst wall.

The wound was closed with dermal sutures after placing a rubber drain. Healing was prompt and the patient was discharged from the hospital.

Microscopic examination of the cyst wall showed it to be composed from within out of columnar epithelium, vascular loose connective tissue and striated muscle fibers. In places the epithelium was absent, possibly due in part to trauma in removing the cyst and in part to inflammation. The fibrous tissue was more compact toward the lumen and looser in structure peripherally. This layer was very vascular, the vessels increasing in size toward the periphery. Positive evidence of inflammation was shown by infiltration of the tissue spaces by fibrin and leucocytes.

October 9, 1931, the patient returned complaining of swelling beneath the tongue and beneath the lower jaw on both sides with some pain on the left side.

On examination a distinct mass could be seen and palpated on each side beneath the tongue, and a mass could be felt in the submaxillary gland region on each side. These swellings were soft, and on pressing on them the masses beneath the tongue appeared to increase in size. Pressure on the mass on the left side caused abundant salivary secretion to run from the opening of the submaxillary duct and diminished the size of the swelling on this side.

October 20, 1931, a small Bowman lacrimal probe was passed into the left submaxillary duct with difficulty, and the duct laid open by cutting down on the probe, making an incision about one and one-fourth inch in length.

The patient was promptly relieved and the swelling on this side had not recurred when the patient was seen last.

Aspiration of the swelling on the right side by an external needle puncture revealed a secretion which resembled saliva. Thinking that possibly the swelling on this side might be due to a remnant of the submaxillary gland tissue producing a similar condition to that on

the left side through stenosis of the submaxillary duct, a probe was passed into the right submaxillary duct November 2, 1931, and the probe cut down on. The incision opened up a good sized cyst cavity into which the finger could be introduced far enough down to palpate the hyoid bone. A thick fluid exactly like that found on removal of the cyst at the first operation was found. The cyst refilled in a few hours and did not diminish in size.

November 18, 1931, a transverse incision was made over the cyst, and the cyst then dissected out. It was found to be very adherent to the skin and surrounded by much scar formation. The sac extended from the angle of the jaw above on the right side downward across the midline to the left submaxillary gland, which, however, it did not involve. A small extension of the cyst evidently representing the duct ran upward medially toward the floor of the mouth. Traction on this portion indented the papilla. In making this maneuver the duct was torn off. An eye probe was passed through the remnant of the duct into the mouth, emerging at the papilla, where it was grasped by an assistant. The distal end of the duct was sutured to the eye of the probe. By pulling the probe out through the mouth, the assistant then evaginated the duct, and cut it off at the papilla. Bleeding vessels were ligated and the incision closed with dermal sutures, after introducing a rubber drain in each angle of the wound. Healing was prompt and convalescence was uneventful.

Grossly no gland structure was seen in the specimen removed; however, on histologic examination a small area of normal salivary gland structure was found, adjacent to which there were tubules which had undergone various stages of cystic dilatation. The epithelium lining these tubules varied from cuboidal to flat squamous like cells.

In this case the cyst was evidently not of branchial or thyroglossal origin, but arose as a retention cyst of the submaxillary salivary gland through obstruction of the submaxillary duct. Due to distention and pressure of the cyst contents, the salivary gland structure underwent atrophy, leaving a cyst with only a small island of normal gland structure. A similar process would have occurred probably on the left side had not the stenosis of the duct been relieved.

COMMENT.

The terms branchial and thyroglossal cysts and fistulæ, to which some writers object, would seem more desirable than the terms lateral and median or midline, which are proposed as substitutes, for

the reason that all thyroglossal cysts, either above or below the hyoid, are not in the midline.

It has been stated that complete thyroglossal fistulæ do not occur; [13 and 14] however, in view of the findings in case 4, it would appear that, although complete fistulæ may be rare, they do occur.

55 East Washington St.

REFERENCES.

1. Young, A. H., and Robinson, A.: Text Book of Anatomy, Cunningham. William Wood & Co., 1921.

2. Hyndman, O. R., and Light, J.: The Branchial Apparatus, Etc. Arch. of Surg., 19:410-52, 1929.

3. Wenglowski, R.: Ueber die Halsfisteln und Cysten. Arch. f. Klin. Chirurg., 98:150-208, 1912.

4. Patterson, N.: Thyroglossal Cysts and Allied Conditions. J. Laryng. and Otol., 43:513-26, 1928.

5. Gilman, P. K.: Cysts and Fistulæ of the Thyroglossal Duct. Surg., Gyn. and Ob., 32:141-9, 1921.

6. Kostanecki and Mielecki: Die angeboren Kiemenfisteln des Menschen. Virch. Arch. Bd., 120 u. 121.

7. Coplin, M. L.: Branchial Cysts and Fistulæ. Philadelphia Med. J., 7:178-82, 1901.

8. Selig, M. G.: Cervical Fistula Following Suppuration in a Patent Thyroglossal Duct. Surg. Gyn. and Ob., 4:623-76, 1907.

9. Cutler, E. C., and Zollinger, R.: The Use of Sclerosing Solutions in the Treatment of Cysts and Fistulæ. Am. J. Surg., 19:411-418, 1933.

10. Schwers, H.: Kyst Branchiogène du cou; errements du diagnostic recédive postopératoire Guérison par injection modificatrice. Liège Médicale, No. 6, February 3, 1933.

11. Wangensteen, O. H.: Differentiation of Branchial from Other Cervical Cysts by X-ray Examination.

12. Sistrunk, W. E.: Technique of Removal of Cysts and Sinuses of the Thyroglossal Duct. Surg. Gyn. and Ob., 46:109-12.

13. Meyer, H. W.: Congenital Cysts and Fistulæ of the Neck. Annals of Surgery, 95:1-26, 1932.

14. Idem., 95:226-247.

LX.

PRESENT STATUS OF AUDIOMETRY.

J. H. Hulka, M. D.,

Long Island City.

I. Introduction.

Relations of audiometry to practical medicine.

To hear means to perceive through the ear the vibrations of the acoustic medium and to appreciate the frequency of the vibrations, as the pitch of the sound and the intensity of the vibrations as loudness.

The relations of the pitch of the sound to the number of vibrations per second are well known. The ear very well distinguishes different tones, and the technic of measurement of the number of vibrations in one second is rather simple.

The relation of the intensity of acoustic vibrations to the loudness of the perceived sound cannot be determined with equal simplicity. Difficulties are not only in measurement of the intensity of the hearing stimulus but also in the determination of loudness.

The ear does not appreciate loudness according to a definite scale like it appreciates pitch. In perception and appreciation of loudness there is no such definite step similar to octave, which is clearly distinguished whenever the frequency is duplicated. Also, to measure the intensity of the hearing stimulus is physically and technically more difficult than to measure its frequency.

Frequency of the hearing stimulus is given by the number of acoustic vibrations acting upon the drum membrane within one second. Intensity of the hearing stimulus is the amount of energy contained in the acoustic vibrations, acting upon 1 cm^2 of the drum membrane in one second.

Frequency of the hearing stimulus is directly determined by the source of the sound and does not change with the acoustic medium or with the distance of the source from the ear. Intensity of the hearing stimulus is not determined by the sound source alone, but depends also on the physical properties of the acoustic medium—elasticity, density, etc.—and changes with the distance of the source from the ear.

The aim of audiometry is to measure the intensity of the hearing stimulus and to bring the results of such measurements into relations with the loudness of the perceived sound. Therefore audiometry is interested in both the physical properties of acoustic vibrations and in psychophysiologic phenomena of audition.

The interest of practical medicine in audiometric research is centered around physiologic and pathologic conditions, which determine the appreciation of the sound intensity of certain sound frequencies. The most frequent disturbance of the hearing faculty is, namely, hypacousis, or lowered perception of the intensity of the sound, which disturbance may be more apparent either in small frequencies (low tones) or in large frequencies (high tones). The interest of practical medicine in audiometry, however, is not limited to otiatry alone. The acoustic nerve and its initial ramifications are very delicate apparatus, which very early reacts to different toxic and systemic influences. As far as such reactions are ascertainable by the measurement of hearing, audiometry belongs also in the field of interests of general medical diagnosis.

Audiometry is not a new medical discipline. The perception of the intensity of sound was investigated for the first time by Friedrich Bezold, who isolated certain frequencies by constructing tuning forks sounding with "pure" tones, and produced in the air hearing stimuli of certain initial intensities by striking the tuning forks with an impact of a constant force. The intensity of so produced hearing stimulus diminishes with the number of seconds. Such diminishing intensity will act as a sufficient hearing stimulus longer, if perceived by a healthy ear, and not so long, if perceived by an ear which is hard of hearing. Deficient ear requires a stimulus of larger intensity in order to perceive sound of minimum loudness than a healthy ear. By his method Bezold was determining the threshold intensity of the hearing stimulus, and this intensity he measured by the seconds of audibility.

The threshold intensity of the hearing stimulus still remains the main interest of medicine in investigation of the hearing disturbances. It is irrelevant for medical purposes, whether the difference of thresholds between a healthy ear and a hard-of-hearing ear is determined in seconds or in units of some other kind. However, the routine application of Bezold method in otology is practiced with many an inaccuracy, which is objectionable from the standpoint of scientific exactness. In the Bezold method of hearing tests the intensity of the sound waves acting upon the drum membrane depends, namely, from many factors, such as the metal from which the tuning fork is constructed, energy and point of the impact, distance of the

tuning fork from the ear, position of the prongs before the meatus, etc. These technical details of tuning fork tests are neither standardized nor rigorously observed, being too exacting in the routine practice. Physicians are furnished with tuning forks made from different materials; a blow with a hammer is supplanted by impacts of other kinds; the fundamental tone of some tuning forks is accompanied by overtones; high-pitch forks sound only for a short period, a circumstance which increases the observational errors, etc. In several tuning fork tests the audibility of the tuning fork for the pathologic ear is being compared with the audibility of the physican's ear, the latter being essentially the basic unit of measurement. Individual variations in the hearing acuity of healthy ears belonging to different otologists shorten or prolong such a basic unit of measurement.

In spite of such inaccuracies and imperfections, the Bezold method of measurement of intensity perception has furnished us with numerous clinical and physiologic observations and during the last decade became one of the basic principles for the construction of physically and technically perfected instruments—audiometers.

TYPES OF AUDIOMETERS.

Several types of audiometers have been constructed. The type which in its applied principle is most similar to Bezold tuning forks is the audiometer 2A, constructed in Bell Telephone Laboratories for the Western Electric Co. in New York.

This instrument produces eight pure tones, the frequencies of which are 64, 128, 256, 512, 1024, 2048 and 8192 cycles—double vibrations—per second. The musical designations of these tones are C, c, c_1, c_2, c_3, c_4, c_5, c_6.

The tones are produced by electric currents, the oscillations of which are regulated by a vacuum lamp. The currents, oscillating in any of the above frequencies, are conducted into a telephone receiver of a special construction, where they produce vibrations of a resonant membrane. The pitch control keys are constructed so that only one tone can be produced at one time.

When the receiver is applied to the ear the vibrations of the resonant membrane produce in the closed space between the receiver and the tympanic membrane certain periodic air-pressure changes, their average magnitude, expressed in bars (1 dyne per 1 cm^2) being in constant mathematical ratio to the voltage of the electric current which produces the vibrations of the resonant membrane. The intensity control dial contains a scale designating the loudness of percep-

tion in sensation units. Consequently, the instrument is constructed for physiologic measurement of hearing and not for physical measurement of the intensity of the sound source and of the hearing stimulus. The physiologic measurement can be, however, transposed into terms designating the physical measurement of the hearing stimulus or of the sound source, because the respective constants are known.

Two types of receivers can be attached to the audiometer 2A. One for the measuring of sound conducted to the drum membrane by the meatus. The other for the measuring of sound conducted to the cochlea through the tissues of the skull. Audiometer 2A is designed for the practical needs of medicine as they have developed from the experiences with Bezold tuning forks.

Nearest to the type 2A is audiometer 1A, which is an enlarged and more detailed instrument. It produces 20 tones in semi-octaves and one-third octaves, from 32 to 16,384 cycles. This type is capable of producing higher intensities of the several frequencies. It is intended to serve for audiometric research.

Audiometers 5A and 3A are designed for the testing of the most important function of the hearing, viz., hearing of the speech. 5A is supplied with energy from the street current, while 3A, 2A and 1A work on dry cells or storage batteries. Types 5A and 3A test hearing of a composite tone of multiple frequencies, similar to those contained in the sounds of speech. They are used for quick hearing tests of employes in transport and industry.

Type 4A is constructed for group tests of hearing of school children. It can be connected with forty receivers. Gramophone record dictates into the receivers a series of numbers, and the children write down the dictation. The loudness of dictation can be varied and by the proportion of omitted or misunderstood numbers the percentage of hearing loss is determined.

Besides these audiometers constructed in the laboratories of the Western Electric Co. several other audiometers for research work are in existence. The audiometer constructed by Kranz can gradually increase the frequency of the tone from 128 to 6000 double vibrations, and this sliding tone can be produced on several intensity levels. The audiometer of Knudson is a combination of both systems. From 64 to 8192 the tones are produced in octaves. From 7000 to 20,000 continuous frequency increase is available. In all these audiometers a telephone receiver is used as the source of the sound.

II. Mathematical and Physical Principles of Audiometry.*

Measuring of the Intensity of Hearing Stimulus.

The intensity of hearing stimulus was defined as the amount of energy contained in the acoustic vibrations acting upon 1 cm.^{2} of the surface of the drum membrane during one second.

The acoustic vibrations cause periodic condensation and rarefaction of the acoustic medium, which, when not disturbed by vibrations, produces a certain pressure on the drum membrane. Under ordinary conditions of hearing the acoustic medium is formed by the air, and its pressure against the drum is given by the atmospheric pressure. By producing a sound wave in the air we produce periodic changes of the atmospheric pressure against the drum. Such changes are measurable. Without going into the technical and theoretical details of this measurement, we shall mention only the principal equations.

Maximum increase p of the pressure produced by a plane sound wave in a small enclosed space filled with gaseous acoustic medium having an initial pressure of p_0 is given by equation

$$p = p_0 a. v. \qquad (1)$$

where a is velocity of propagation of the wave and v is the velocity of the vibrating particles of the given acoustic medium. All these values can be determined; the last mentioned (v) is figured out from the velocity of an electrodynamically driven piston, which produces the sound wave in the given enclosed space. Such a small enclosed space with a "piston" producing a sound is formed by applying a telephone receiver tightly against the ear. Then the velocity v, mentioned in the above equation, can be figured out from the amplitude and the frequency of the resonant membrane.

The pressure changes produced in the enclosed space between the telephone receiver and the drum membrane are called in telephony the sound pressure. The sound pressure, however, is not determined by the maximal pressure changes caused by the acoustic waves but by the average (root—mean—square) pressure changes P. The sound pressure P is measured in the units of the barometric pressure called bars (1 bar = pressure of one dyne against 1 cm.^{2} One dyne is approximately the force exerted by a milligram weight under the influence of gravity. Atmospheric pressure measures approximately 1,000,000 bars.)

*The author thanks the Bell Telephone Laboratories for the revision of this part of the essay.

The sound pressure P is in a definite relation to the intensity I of the sound waves, given by the equation

$$I = \frac{p^2}{p_0 \cdot c} \text{ (ergs per sec. per. cm}^2\text{)}. \qquad (2)$$

where c means velocity of the sound in the air in cm. per sec., and p_0 (atmospheric pressure) is expressed as density of the air.

This equation shows that the measuring of the intensity of the telephonic hearing stimulus is based on the measuring of the sound pressure; the latter is determined by the amplitude and by the frequency of the resonant membrane. It was already mentioned that the telephone receiver of the audiometer has been constructed so that the sound pressure is directly proportional to the voltage at the terminals of the receiver because the voltage is better measurable than the amplitude. The numerical relation between the voltage of the receiver and the sound pressure is different for different frequencies but constant for all intensities of the same frequency.

The intensity of the hearing stimulus produced by the receiver of the audiometer is, as has been shown, perfectly determined and measurable in physical units of force and energy.

MEASURING OF SUBJECTIVE QUANTITIES.

It was stated that the aim of audiometry is the determination of the relations between the intensity of the hearing stimulus and the loudness of the perceived sound.

To determine such a relation metrically, we have to measure not only the intensity of the hearing stimulus but also the loudness of the preception.

Hearing and its loudness is a subjective experience and though in subjective experiences we can very well distinguish their quantities (small pain, big pain and sim.), the measuring of such psychologic quantities lacks the exactness with which the physical quantities are measurable.

The method of measurement of subjective quantities was originally based on determination of a series of minimum perceptible changes in the quantity of a perception of the same kind, and the quantity of the measured perception was then expressed by the number of units in the found progression.

An example will explain this method better than statement of its general principle:

Suppose that we have to measure the quantity of the pressure sensation caused by the weight of 1 kg. of sand on our palm. As stated, we shall measure it by the number of members in the progression of minimum perceptible changes of the pressure sensation.

Such progression can be elicited as follows:

Let us hold on the palm of our hand a vessel of small and mathematically negligible weight. Let us close our eyes and have somebody to pour sand into the vessel. It will be necessary to place into the vessel a certain amount of sand, weighing (a) grams, to produce the first minimal sensation of pressure against the palm. By pouring this weight of sand into the vessel we produce the threshold sensation of pressure.

Holding further the vessel with (a) grams of the sand on the hand and pouring in additional sand we produce, after having added a certain amount of sand, the first minimum sensation of change of the pressure. This way was elicited the first *"unit"* of pressure sensation. Noting down the number of grams of the sand now contained in the vessel, we continue filling the vessel until we produce the second, third, fourth, etc., minimum sensation of the change in the pressure against the palm, i. e., until we elicit two, three, four, etc., "units" of pressure sensation. In the process of gradually placing 1 kg. of sand on the palm we have produced the feeling of y minimum changes in pressure. The magnitude of pressure sensation against the palm holding 1 kg. of sand is then y "sensation units."

THE RELATION OF THE QUANTITIES OF STIMULI TO THE QUANTITIES OF SENSATIONS.

It is evident from the above example that there is a certain mathematical relation between the number of "units" of the subjective sensation and the quantities of the stimuli, i. e., the weights of the sand in our experiment, which are necessary to produce such a progression of minimum perceptible changes.

In our experiment we could find that each weight which elicited the following sensation unit is certain multiple of the weight which elicited the just preceding sensation unit.

Quantitative relations between the stimulus and the sensation were investigated for the first time by Weber,[2] in Leipzig, and the definition of minimum perceptible change as "sensation unit" was later introduced by Theodore Fechner.[3]

Weber and Fechner thought that between any two stimuli which elicit any minimum sensation of change was a constant relation. This

opinion was expressed by Fechner as a psycho-physical law: The sensation increases in arithmetical proportion as the stimulus increases in geometrical proportion.

According to the Fechner law, if we designate the quantities of the successive stimuli as (a), (b), (c), (d), etc., then (a). r = (b), (b). r = (c), (c). r = (d), etc.

The quantity of stimulus x after y times produced change in the above experiment is, according to Fechner, given by an equation.

$$x = (a).\, r^{y}. \qquad (3)$$

where (a) means the threshold stimulus, r means the quotient obtained by dividing the following by the preceding stimulus, and y means the number of Fechner's sensation units.

THE MEASURING OF LOUDNESS IN TELEPHONY: BEL.

Weber's original investigation of psychophysical relations concerned the tactile and the auditory senses. The ear is essentially a modification of the tactile organ. Vibrations, the frequencies of which are less than about 12, give rise to a tactile sensation of tremulous character. Vibrations of high intensities cause a sensation of pain. Therefore the example mentioned in the previous chapter can very well serve as an illustration of the principles of measurement of auditory sensations.

The Fechner method, however, is not satisfactory in practical application for measuring of loudness in telephonic transmission of human speech. Speech contains many compound sounds and minimum perceptible differences of the loudness of speech cannot be determined with sufficient precision. Besides that, we are not so much concerned about minimum differences of loudness in telephony; we are interested in much larger changes.

Therefore, by an international agreement, for the purposes of telephony a gradation of loudness has been accepted, which is based on multiplication of the stimulus intensity by ten. Such grades of loudness have been accepted as units of measure for the auditory sensation, and are called bels. (After the name of the inventor of the telephone, Alexander Graham Bell, 1847-1922.)

If the Fechner law is valid, then a hearing stimulus, which increases in geometrical progression, i. e., 10 times, 100 times, 1000 times, etc., will evoke a hearing sensation, the loudness of which increases by 1, 2, 3, etc., equal steps.

The common ratio of a geometrical progression of stimuli in bel-grades equals 10, and the quantity of a hearing stimulus (given by the intensity of the acoustic vibrations I) after b times accomplished intensity change (always in a ratio of 10:1) is given as follows:

$$I = k.\ 10^{b}. \qquad (4)$$

where b means not only the number of accomplished changes but also loudness expressed in bels, and k means the intensity of the stimulus necessary for the eliciting of "zero" or "reference" loudness, from which the measuring begins. Equation (4) is derived from equation (3).

If for reference we accept a loudness, produced by a stimulus intensity of 10^{-16} Watt/sec. cm.^{2}, and mark it in the diagram (No. 1) by a zero on the axis of ordinates, and if we mark one-bel-larger loudness on the same axis by a line 5 mm. high, then the loudness of the graphically represented equation (4) for a tone of 1024 cycles per second will increase with the scale B of our diagram.

The intensity of stimulus necessary to produce a loudness of one bel is represented on the diagram by a column 1 mm. in length. The amount of this intensity in Watts is given by the number 10^{-15} in the scale C.

The diagram shows that the loudness of tone c^{5} (1024 double vibrations) can be graded in 13 bel units before it reaches its upper threshold, where the hearing sensation changes into a sensation of pain.

To effect such a gradation we must have at our disposal a stimulus intensity changeable in enormous limits. To elicit a loudness of one bel we need an intensity represented by one millimeter. To evoke a loudness of 13 bels we need an intensity represented by an astronomical dimension of 1,000,000 km.

AUDIOMETRIC SENSATION UNIT: ITS RELATIONS TO THE MINIMUM PERCEPTIBLE CHANGES OF LOUDNESS AND TO DECIBEL.

In audiometric research undertaken for technical or medical purposes we have to investigate occasionally a finer gradation of loudness than that given by the telephonic unit of one bel.

Therefore audiometry, which developed from the researches in telephony, accepted one-tenth of the bel as the unit of loudness and this unit is called Sensation Unit, with a designation S U.

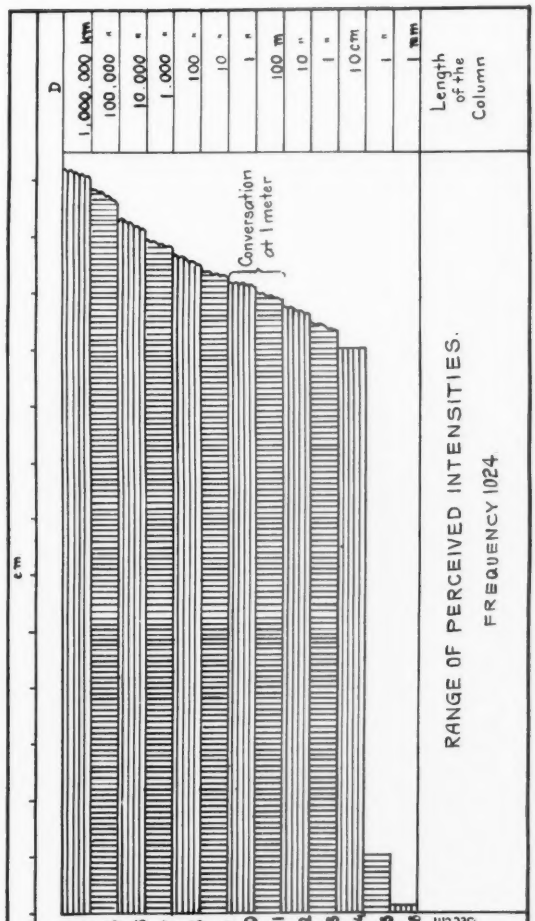

RANGE OF PERCEIVED INTENSITIES.
FREQUENCY 1024
Conversation at 1 meter
Length of the Column
1,000,000 km
100,000 "
10,000 "
1,000 "
100 "
10 "
1 "
100 m
10 "
1 "
10 cm
1 "
1 mm
Sensation Units Decibels
Bels
Intensity in Watts/sec cm²
A B C D
cm

Fig. 1.

When loudness is measured by an audiometer, constructed for medical purposes, its magnitude is expressed by the number of Sensation Units above the threshold loudness of the tested tone, the threshold loudness being designated as zero.

The change of intensity of the hearing stimulus, which is necessary to increase the loudness of the tested tone by one Sensation Unit is called one decibel.

The stimulus intensity of a tone which is SU Sensation Units loud is given by an equation derived from the relation of the Sensation Unit to bel and from equation (4):

$$I = (a_f) . 10^{SU/10}. \qquad (5)$$

where (a_f) means a stimulus intensity necessary to produce the threshold loudness of a tone characterized by frequency f.

If the audiometric Sensation Units were identical with Fechner's "sensation units" for the auditory sense (the latter being defined as the minimum perceptible change of hearing sensation), then the quotient r in equation (3) would equal about 1·26 (more exactly $\log r$ would equal 0·1).

Quotients for minimum perceptible changes of the auditory sensations were investigated by R. R. Riesz[4] by the method of beats. Riesz found (like previously Hecht,[5] Charpentier[6] and other investigators of other sense organs) that the law of minimum discernible for the hearing organ is far more complicated than Fechner's formula.

Riesz's researches discovered, however, that with the exception of the lowest audible intensities, the quotient is never larger than 3·16, which is the ratio of two intensities, producing tones differing in loudness by 5 audiometric Sensation Units.

That is why audiometer is calibrated in steps of 5 Sensation Units. It gives the instrument a mathematical assurance that such gradation of loudness will be distinguished by the human ear in all tests, with the exception of lowest intensities. But even in such threshold-approaching tests the change of 10 Sensation Units will be appreciated by the human ear.

Audiometic Sensation Unit is an artificial mathematical unit, which is determined and measured by the intensity of stimulus. It is not a natural, psychophysical unit and it does not correspond with the minimum perceptible changes. Within the limits of five Sensation Units are almost all minimum perceptible changes of the loudness

sensation. The majority of such minimum perceptible changes are elicited by changes of the hearing stimulus, which are approximately identical with those necessary to produce an increase of loudness by one Sensation Unit.

Theoretically the audiometric Sensation Unit is a unit of measure of loudness, which equals the increase of loudness elicited by multiplication of any hearing stimulus by 1·26. Such a small increase of loudness, however, is in many instances unreal, imperceivable and only mathematically assumed.

The relations of minimum perceptible changes of loudness to the number of sensation units for a tone of 1000 cycles are represented by the scales A, B, C and D in diagram 2. Near the threshold (scale A and B) we have to change the intensity by approximately 3·1 decibels to be aware of the first change of loudness. If the tone of 1000 cycles is of conversational loudness (scale C), we are able to perceive a change as soon as the stimulus intensity is increased by about 0·46 of a decibel.[7]

For tones of other frequencies these relations are similar, though somewhat different in detail.

Diagram 2 also indicates that decibel is mathematically identical with Sensation Unit. Should we mark the intensity of the stimulus on the horizontal axis in decibels, and the loudness of the tone on the vertical axis in Sensation Units, we would obtain a straight line.

REFERENCE LOUDNESS AND REFERENCE INTENSITY.

It is evident from the loudness scales of diagrams 1 and 2 that the measurement of loudness has to start from a certain reference point. Such a natural reference point is a loudness of a threshold perception. That is how the referencee points for the graduation of the control dials of the 2A and 1A audiometers were selected.

Consequently the reference loudness of each frequency tested by the audiometer is produced by a different amount of stimulus intensity.

The analysis of decibel in the previous chapter shows that the stimulus intensity can just as well be expressed in decibels as in energy units, Watts or ergs. However, if we want to express the stimulus intensity in decibels we again have to select a certain point as reference intensity. This can be seen from the scale B of diagram 1.

Audiometric calculations are usually quoted in decibels, which are essentially "relative" units, meaning a change from or a relation

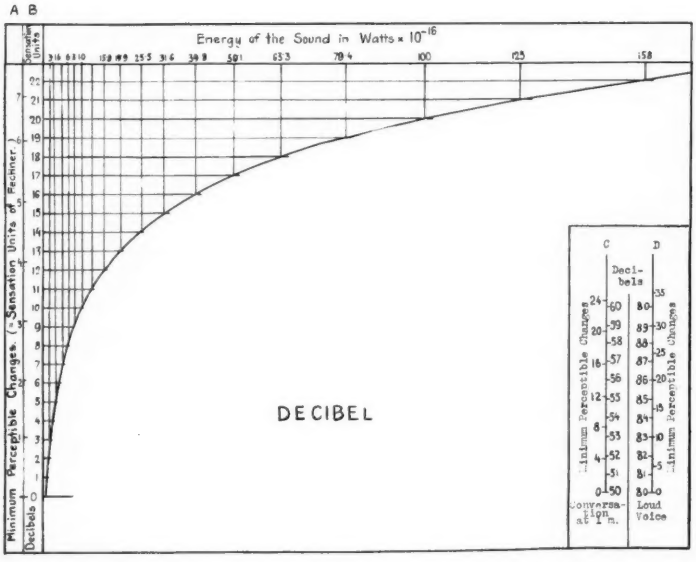


Fig. 2. Decibel and its relation to the minimum perceptible changes, to the energy in Watts, and to the Sensation Units for a reference tone of 1000 double vibrations.

to a certain standard, viz., the reference intensity. The American Standards Association suggests for reference intensity

$$I_0 = 10^{-16} \text{ watt per sec. cm.}^2$$

This intensity was used for the reference point of the diagram 2. Substituting this reference intensity for k in equation (4) we obtain

$$I = 10^{-16}\, 10^{b} \text{ watt / sec. cm.}^2 \qquad (6a)$$
$$= 10^{b-16} \text{ watt / sec. cm.}^2 \qquad (6b)$$

Similarly we can write equation (5) by using instead of SU the mathematically identical value of decibel (db)

$$I = 10^{db/10 \,-\, 16} \text{ watt / sec. cm.}^2 \qquad (7)$$

Equations 6b and 7 serve for reduction of bels to Watts and it is with advantage to write them in their logarithmic forms:

$$log_{10} I = b - 16, \text{ and} \qquad (8)$$
$$10\, log_{10} I = db - 160 \qquad (9)$$

where I is intesity given in Watts, and b and db are intensities given in bels and decibels.

With the standardization of reference intensity also a standardization of reference loudness was proposed,[3] so as to make reference loudness mathematically identical with reference intensity. That means: Reference loudness of a certain tone should be a loudness which is produced by a stimulus of corresponding frequency, having an intensity of 10^{-16} Watt per sec. per cm.^{2} Such reference loudness can be used with advantage for expression of mathematical relations between intensity and loudness, and is so used also in this essay. On the other hand, such selection of reference loudness does not contribute to the clear and practical understanding of the conception of loudness. Diagram 4 shows that with standardized reference loudness the threshold loudness of c^1 equals 40 Sensation Units, while threshold loudness of c^3 equals 8·129 Sensation Units.

The threshold perceptions of c^3 and c^1 are more nearly equally loud than 37 degrees different. For this reason it is more practical to leave reference loudness at the threshold of the frequencies (like it is at the audiometric dials), whenever we are concerned with relations of loudness to any other value but the intensity of the stimulus.

DEFINITION OF BEL AND DECIBEL.

Before attempting to define bel and decibel as units of intensity measure, we shall say a few words about logarithms for the benefit of readers who are not daily occupied with mathematics.

Logarithm of a number is the index of the power to which a base must be raised to give the number. If the base is 10, then logarithm of 100 is 2. This is written

$$\log_{10} 100 = 2, \text{ because}$$
$$100 = 10^2$$

Like the number 2 in the last equation, bel (b) occurs as an exponent of 10 in the equation 7a. Therefore, it is a number which means or designates a logarithm, if the base is 10.

If bel is unit of measure, then $b = 1$, the 1 being a number designating a logarithm.

Consequently, we can define bel as unit of measure of sound intensity, which equals the logarithmic value of the number 1. Similarly decibel is a unit of measure of sound intensity, which equals the logarithmic value of the number 0·1.

Such definitions mean that numbers which designate intensity in bels or decibels are not ordinary numbers. They are logarithmic

numbers. Therefore the amount of intensity given in bels is at the same time a logarithmic expression of intensity given in Watts.

A plastic explanation of these definitions would be as follows: Assuming that sound intensity is something measurable with a rod similar to a meter or to a yard, then the numbers 1, 2, 3, 4, etc., designating bels, would not be placed on such a rod in equal distances, like similar numbers on a yard-rod or meter-rod; they would be displaced in proportions of 1:10:100:1000, etc.

RELATIONS BETWEEN THE SOURCE, THE STIMULUS AND THE PERCEPTION.

The fundamental measuring in audiometry is the measuring of that sound pressure which elicits a threshold sensation of hearing in a normal ear.

This sound pressure is given in the first row of table 1 for the octaves of tone c. It is effected by applying to the ear an audiometric receiver through which a certain voltage of oscillating electric current is passing So elicited threshold loudness is the reference loudness of audiometer.

Therefore the fundamental equation of applied audiometry is:

$$V_{of} = k_f \, P_{of} \text{ volt,}$$

where V_{of} means voltage of the receiver necessary for eliciting threshold loudness of a certain tone, P_{of} means pressure of the sound against 1 cm.^{2} of the tympanitic membrane (in bars) and k_f is a voltage constant of a receiver for the respective frequency.

Threshold intensity of the stimulus delivered from the receiver to the ear is given by equation (2), using the value of 41·5 for air at 20 C°:

$$I_{of} = \frac{P^2_{of}}{41{\cdot}5} \, 10^{-8} \text{ watt / sec. cm.}^2$$

The values for I_o for frequencies of audiometer 2A are given in the second row of table 1.

The energy of the receiver is in a constant relation to the intensity of a stimulus of each frequency. Therefore we can write:

$$E_{of} = K_f \cdot I_{of} \text{ watt.}$$

Constants of the receivers depend on their construction. They determine the volts, amperes and ohms applied in the production of the several tones by the audiometer.

TABLE 1.

THRESHOLD VALUES OF SOUND STIMULUS.

Frequency	64	128	256	512	1024	2048	4096	8192	16384
Threshold sound pressure[9] in bars, P_{of} (times 10^{-4})	1200	210	39	10	5.2	4.1	4.2	25	1300
Threshold intensity[9] I_{of} in watts (times 10^{-16})	350.000	10600	360	24	6.5	4.0	4.2	150	410.000
Threshold intensity in decibels I_{db}	55.4407	40.2531	25.5630	13.8021	8.1291	6.0206	6.2325	21.7609	56.1278

Intensities of threshold stimuli in decibels I_{db} if reference intensity

$$I_0 = 10^{-16} \text{ watt / sec. cm.}^2$$

are given in the third row of the table 1. Their equation is:

$$I_{db} = 10. \log_{10} I_{of} + 160 \text{ decibels}$$

The sound pressure of a tone, which according to the dial reading is SU, Sensation Units loud, is given by equation

$$P_{SU} = P_{of} \cdot 10^{(SU-80)/20} \text{ bars}$$

Stimulus intensity of a tone, which according to the dial reading is SU Sensation Units loud is given by equation:

$$I_{SU} = I_{of} \cdot 10^{(SU-160)/10} \text{ watt / sec. cm.}^2$$

Voltage on the terminals of the receiver, which is necessary to produce loudness of SU Sensation Units is found from equation:

$$V_{SU} = k_f \cdot P_{SU} \text{ volt.}$$

And energy, which passes through the receiver when the dial is turned to SU Sensation Units, is given by equation:

$$E_{SU} = K_f \cdot I_{SU} \text{ watt.}$$

These equations show that Western Electric Audiometer complies with the principal requirements of audiometry, namely, to bring into definite numerical relations the three principal factors taking place in the phenomenon of audition: The source of the sound, the hearing stimulus and the perception of the sound. The instrument complies with them because the relation between the energy of the source and the intensity of the stimulus is measurable, if the source is a telephone receiver applied to the ear.

If a different source is used, which is distant from the ear and which is not connected with the ear by a small enclosed space, the relations of the energy of the source to the intensity of the stimulus are much more complicated and difficult to find. That is why audiometers producing sounds by forks or megaphones lack the precision of the audiometers with which a telephone receiver is used as a source of the sound.

III. Principles of Audiography.

Graphic representation of range of hearing stimuli.

Hearing stimuli are acoustic vibrations characterized by certain frequencies and by certain intensities. Both frequencies and intensities can be measured. Therefore it is possible to determine every sound by two measurable values. Sound wave may have an audible

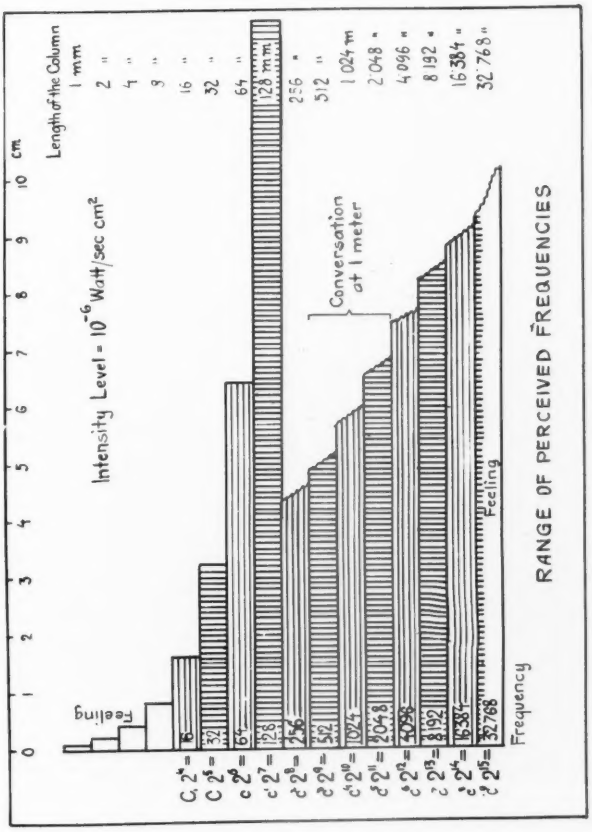

Length of the Column
1 mm
2 "
4 "
8 "
16 "
32 "
64 "
128 mm
256 "
512 "
1 024 m
2 048 "
4 096 "
8 192 "
16 384 "
32 768 "
0 1 2 3 4 5 6 7 8 9 10 cm
Intensity Level = 10^{-6} Watt/sec cm²
Conversation at 1 meter
Feeling
Hearing
Frequency
RANGE OF PERCEIVED FREQUENCIES

Fig. 3.

frequency but it may not be heard because its intensity is too small. If its intensity is too large, it also will not be heard; it will produce a sensation of pain.

The range of frequencies audible on intensity level of 10^{-16} Watt per sec. per cm.^{2} is represented by diagram 3.

Similarly diagram 1 represents range of intensities for a tone of 1024 double vibrations.

The range of all audible intensities and of all audible frequencies could be represented by a field where every point would be determined by two co-ordinates; by an abscissa of intensity and by an ordinate of frequency.

Before starting to draw a diagram of such a field we have to know the maximum dimensions of the graph.

From the diagrams 1 and 3 we can see that to represent all values of frequencies and all values of intensities by intelligible dimensions the graph would have to have maximum dimensions of about 32 meters by 1,000,000 km. Such a graph cannot be designed. It is necessary to find another graphic method to represent the range of hearing stimuli. Such method results from the quantitative relations of hearing stimuli to sound perceptions.

GRAPHIC REPRESENTATION OF RANGE OF SOUND PERCEPTIONS: SOUND FIELD.

Audiometry views hearing as a measurable subjective phenomenon characterized by two quantities: Height of the pitch and degree of the loudness. These two quantities can be graphically represented as two dimensions in the same plane. Each point of such plane then means perception of a certain tone characterized by a certain pitch and by a certain loudness.

For the unit of measure of loudness we have accepted the audiometric Sensation Unit.

For the unit of measure of pitch we can accept any musical interval which is usually well distinguished by the ear. One of the basic musical intervals is the octave, which has its origin in duplication of frequency of the hearing stimulus.

The range of sound perception can be represented by a plane figure with one dimension measured by Sensation Units, and with the other dimension measured by octaves.

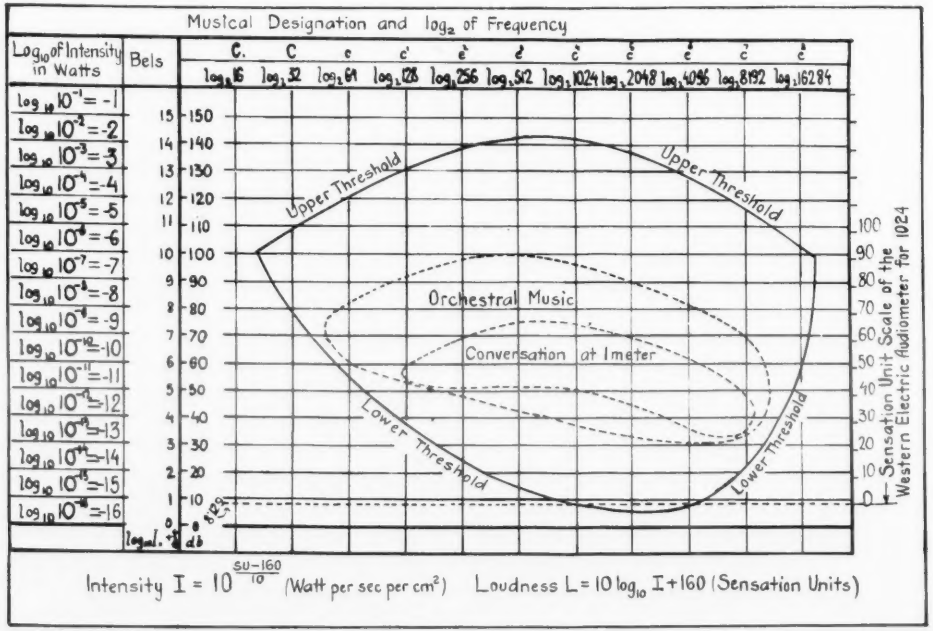


Fig. 4. Sound Field. Coordinates are given by the logarithms of frequencies and intensities. (Curves of the upper and lower thresholds represent averages resulting from a measurement of many ears.[9] Fields of conversation and orchestral music according to Bell Laboratories Record, June, 1934, pp. 314 and 315.)

These measures are represented in diagram 4 by vertical and horizontal scales. Octaves of the tone c are marked on the horizontal scale, Sensation Units on the vertical scale. If for each audible tone we measure out its maximum and minimum loudness, we obtain a plane figure limited by two curves, which represent the upper and the lower thresholds of hearing. Such a figure is called sound field.

Sound field is not only a graphic representation of the range of sound perception; it is at the same time a logarithmic graph of the range of hearing stimuli.

The horizontal scale of Fig. 4 demonstrates that octave is determined by the logarithm of frequency if the base is 2. Similarly the loudness in Sensation Units is determined by logarithm of intensity, if the base is 10, as can be seen on the vertical scale.

HEARING FIELD: AUDIOGRAM OF A NORMAL EAR.

The scale of loudness in diagram 4 was chosen so that loudness has the same mathematical determination as the stimulus intensity given in decibels. It was mentioned in the chapter treating reference loudness that such scale is not quite practical for the comparison of loudness of different frequencies.

To the audiometric control dial are applied scales of loudness which start from threshold loudness of each frequency. By plotting a diagram of sound field on such scales we transform the curve of the lower threshold into a straight line and at the same time the curve of the upper threshold obtains a larger convexity.

In medical audiometry we are most interested in graphic representation of lowered hearing faculty. To illustrate a lowered hearing we have to adjust the graph of the sound field so that minimum sounds perceived by the defective ear are pictured lower than minimum sounds perceptible by a normal ear. Such adjusted graph is given in diagram 5 and is called hearing field, or audiogram of a normal ear.

DETERMINATION OF NORMAL LOWER THRESHOLDS OF LOUDNESS.

Normal lower thresholds of loudness are determined by the minimum intensity which, acting upon a normal ear, elicits a sensation of hearing. Lower thresholds of loudness constitute the acuity curve of hearing. (See the following chapter.)

First we have to define the meaning of "normal" ear. Normality of the hearing organ is not given by any definite physical principle

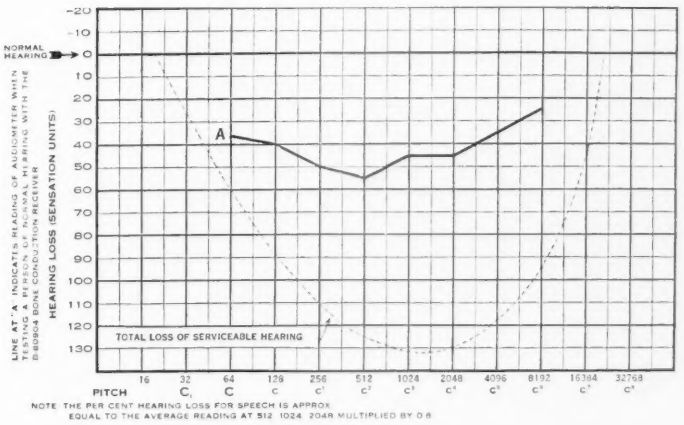


Fig. 5. Hearing Field. Audiogram of a normal ear.

like, for instance, the emmetrophy of the eye. Emmetropic eye, the visual acuity of which is very often referred to as a "normal" vision, is determined physically by its dimensions and by the optical properties of the refracting media. For "normal" acuity of hearing and for "normal" ear we have no such precise physical determination.

With other organs, their normal functions, as far as they are measurable, are defined in many different ways. So the "normal" faculty of kidneys to eliminate nitrogen compounds is determined by the amount of nitrogen found in a certain volume of blood taken from the veins of healthy individuals. Normality defined in this way is usually assigned certain maximum and minimum limits.

Where we are concerned with finding of quantitative differences between the functions of a normal and of a pathologic organ, it is with advantage to define normality as a constant and not as a fluctuating value. For such a definition of normality an average of certain variations is suitable.

For "normal" hearing acuity could be accepted the mean of physiologic variations of hearing acuities of a great number of healthy individuals. Such variations, however, have very broad dispersion, and depend from age, education, mentality, training and from many other factors.

The dispersions will be narrower if we choose only individuals selected according to a certain rule or standard.

Normal thresholds designated by a zero line in the audiogram of a normal ear are a mean of threshold intensities which were found by several independent investigators who performed measurements of eighty-nine ears with good acuities and without traces of pathology in history or on physical examination.[9, 10]

Such audiometric normal is rather arbitrary and deviations from it cannot be assigned definite pathologic meanings unless either the limits or the dispersions of physiologic variations are known. This will be the subject of a later discussion.

THE CURVES OF HEARING ACUITY: THE FIELD OF LOSS OF HEARING.

The routine audiometric tests are performed to find curves of hearing acuity.

"Normal" ear begins to hear a tested frequency if the control dial of the audiometer is turned with zero against the threshold of the frequency. An ear which has a lowered hearing acuity requires turning of the disc by one or more degrees of the scale to the right. Each degree of the scale by which the dial has to be turned to the right means a loss of 5 Sensation Units of hearing acuity. (See diagram 6.)

By plotting the losses of hearing acuity for the tested frequencies on the diagram of the hearing field we receive a curve of hearing acuity. The curve is given by points designating the threshold loudness of the tested ear. (See diagram 7.)

The plane figure A, B, C, D represents the field of the loss of hearing as far as it could be determined by the audiometer 2A. The losses in frequencies lower than 64 and higher than 8192 stay undetermined. The thresholds of extreme frequencies, however, are subject to so frequent and so wide physiologic variations that their values have very little meaning in pathology of the ear. They can be found by the audiometer 1A, which is to serve theoretical and physiologic research.

In clinical examination another method of auditory tests has been established since a long time, namely, testing of a bone conduction. Audiometers 1A and 2A are supplied with bone conduction receivers, and "normal" hearing acuity for the bone conduction receiver is indicated in the hearing field diagrams (audiogram blanks) by the curve A. Deviations of the tested ear from this normal constitute a curve of hearing acuity through bone conduction.

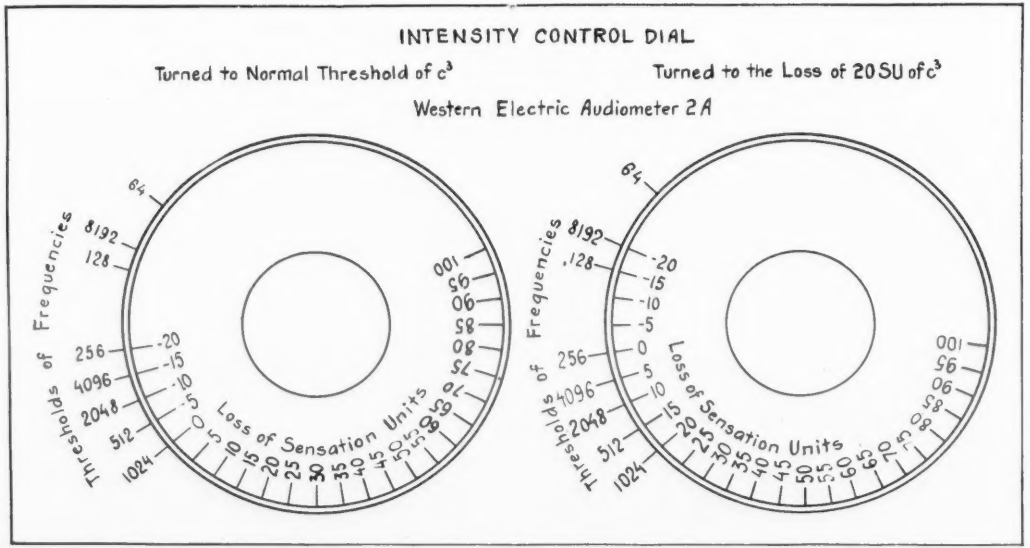


Fig. 6. Intensity Control Dial of Western Electric Audiometer 2A. Receiver sounding with tone c^3 produces, if applied to the ear, a hearing stimulus having an intensity of $6{\cdot}5 \ .10^{-16}$ watts as long as the control disc is in the first position. If the tested ear does not hear the tone, until the disc is turned into the second position, when the stimulus intensity equals $6{\cdot}5 \ .10^{-14}$ watts, loss of 20 SU in the frequency 1024 has been discovered.

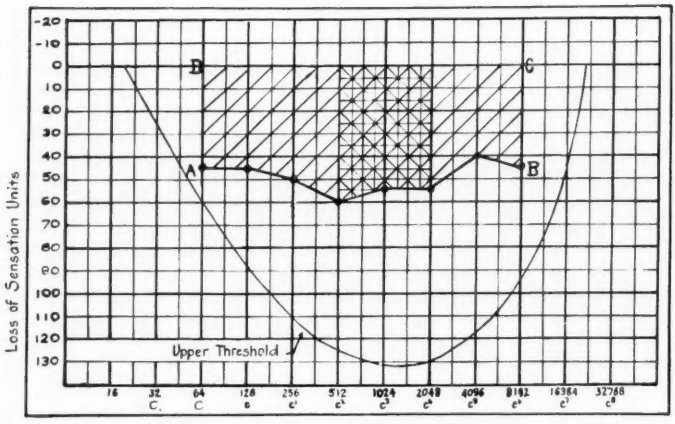


Fig. 7. The Curve of Hearing Acuity. *A B* Acuity curve for air conduction. *A B C D* field of the loss of hearing. Per cent hearing loss for speech = average hearing at 512, 1024, 2048 multiplied by 0·8. (Represented by the double shaded area.)

By testing the ear with the two receivers we obtain two hearing acuity curves: Curve of the air conduction and curve of the bone conduction.

The curve of air conduction is a pure curve of meatal conduction only as long as the loss of hearing is not more than about 40 to 60[11] Sensation Units. With higher degrees of deafness the air conduction receiver causes hearing through the bone. In unilateral deafness the opposite ear may register (through bone conduction) even intensities 20 to 30 decibels above those of the audiometric normal.[12] Therefore to find a true curve of air conduction of the worse ear we have to mask the hearing of the opposite ear with noise whenever the difference between the two tested ears appears to be more than 25 to 30 SU, and if, after the masking of the opposite ear, the tested ear gives a curve lower than 40 to 60 SU below normal, such curve indicates a hearing aided by bone conduction of the tested ear.

To obtain a true curve of bone conduction, masking of the opposite ear should be practiced as a routine procedure. The difference of bone conduction acuity in unilateral deafness hardly exceeds 10 to 20 SU with bone conduction receiver placed first over one and then over the second mastoid process.

The buzzing sound of 5A or 3A audiometer is the best means for masking.[12]

IV. The Air Conduction Curve.

THE AIR CONDUCTION ACUITY OF A HEALTHY HEARING APPARATUS.

To find a meaning of the air conduction acuity for the pathology of the ear we first have to become acquainted with the variations of acuity of hearing in healthy individuals. Acuity of hearing of a healthy individual we shall call physiologic acuity. Practical measuring of variations of physiologic acuity is rather difficult because of the requirement that the hearing apparatus, which can be taken into consideration, have to be found healthy by methods other than acuity tests.

By hearing apparatus we understand three principal anatomic equipments, which function during the phenomenon of audition: 1. The conduction apparatus (the auricle, meatus, tympanitic membrane and ossicles). 2. The perception apparatus (the cochlea with anatomic details and with the ramification of the fibers of the spiral ganglion). 3. Central nerve system (the auditory nerve, its central paths with ganglia and the cerebral cortex.)

To appraise the state of health of this whole apparatus is no easy task. The conduction apparatus of the ear cannot be investigated with the precision of the retinoscopic refraction of the eye. Otoscopy does not allow us to see the cochlea, while ophthalmoscopy shows us the perceiving part of the visual organ. In otology we know of no test comparable to the investigation of visual fields which supplies the ophthalmologist with rather exact information about the condition of the optic tracts and of the cuneal cortex.

Only after postmortem microscopic examination we can decide whether the total hearing apparatus is healthy or diseased. But even such examination will discover only morphologic changes. So-called "functional" pathology will stay without postmortal evidence.

Though it is difficult to prove that the hearing apparatus is healthy, it can be assumed—if there is no indication of aural disease in the history and in the physical examination of the tested individual. We have to admit that such assumption will not be correct in every case; but with the present status of our knowledge of the hearing apparatus such assumed physiologic acuity of hearing is the only measurable physiologic acuity.

The physiologic hearing acuity is to be discriminated in this essay from the "normal" hearing acuity, which has been previously defined as the mean acuity of an arbitrarily selected group of test subjects.

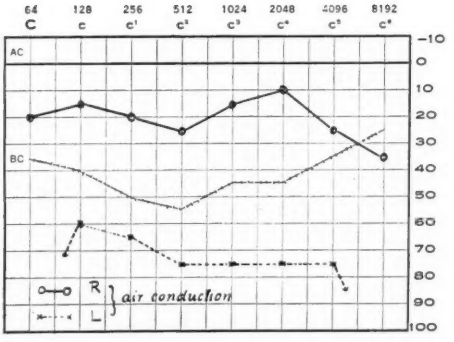


Fig. 8. Very Different Air Conduction Acuities of the Two Ears of the Same Person. Both ears healthy. No explanation found.[13] BC = normal for bone conduction receiver.

VARIATIONS OF PHYSIOLOGIC AIR CONDUCTION ACUITY.

Physiologic air conduction acuity, when measured on a large number of persons, fluctuates within rather wide limits. It can even show a wide difference between both ears of the same individual. A case of this kind has been described by Stacy R. Guild[13] (see diagram 8). Postmortal examination did not furnish any explanation of the difference. (Guild does not mention whether the central paths of this case were examined.)

This, to my knowledge, most detailed study of a case of physiologic variations gives weight to other studies which touch the problem of physiologic variations, without, however, microscopically investigating the hearing apparatus. The several studies which will be mentioned assume the health of the hearing apparatus merely because of clinical elimination of pathology.

Dana W. Drury[14] reports variations of physiologic hearing acuity in children and young individuals. Typical examples of average and variations in a group of children are represented by diagram 9a. The variations fluctuate within limits about 40 SU wide. Groups differently selected present narrower fluctuations. Variations of a group of singers fluctuate within limits 20 SU wide. Similar narrow limits have the variations of trained telephone operators, athletes, pianists, etc. With the training of the hearing not only the limits of the variations become narrower but also the average approaches a higher level.

C. C. Bunch[15] summarized the examinations of 353 persons classified by age groups into a number of "dispersograms," which

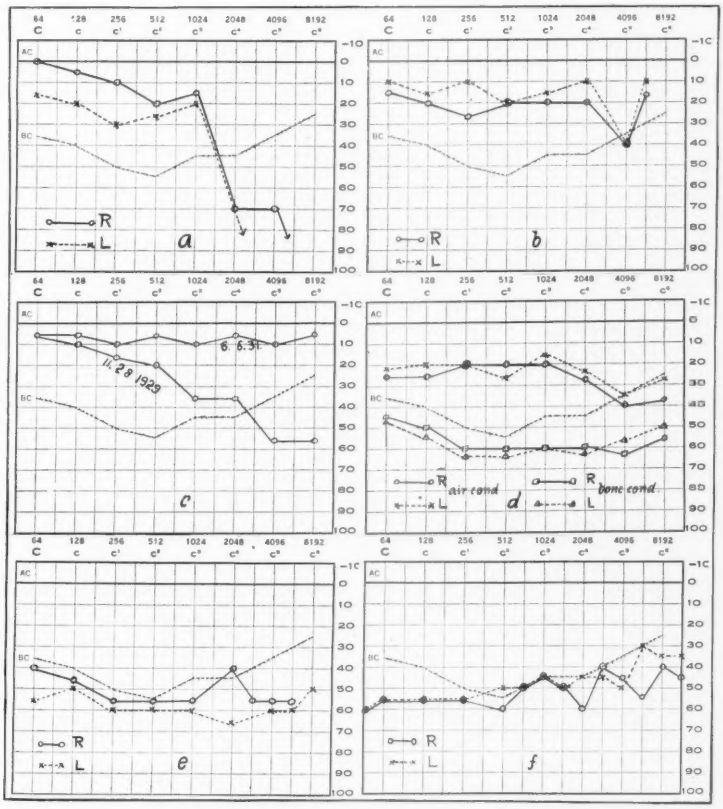


Fig. 9. Averages and Variations of Air Conduction Acuity According to D. W. Drury.[11]

——— Averages.

. . . . . Maximum variations from the average.

- - - - - Normal for bone conduction receiver.

a, 50 public school children. *b*, 50 untrained telephone girls. *c*, students, athletes. *d*, 50 trained telephone girls. *e*, 10 piano students. *f*, 10 voice students.

depict the variations of hearing acuity. The subjects were patients of a surgical hospital department. They were not conscious of any defect of their hearing and were not considered hard of hearing by the people of their surrounding. (It is not evident from the Bunch study that any thoroughgoing otologic examinations of the subjects were performed for the purpose of elimination of pathology.)

Acuity variations of so assumed "healthy ear" present dispersions which vary with the pitch of the tested tone. The limits of variations from 32 to 2048 double vibrations are about 35 SU wide. In higher frequencies the width of the limits increases with the pitch and comprises as much as 75 SU. (See diagram 10.)

The age of the tested individuals has an influence on the average of the hearing acuity, especially so in higher frequencies. Average acuity in octaves higher than c^3 (1024 dv) rapidly decreases after the thirtieth year with an approximate rate of one unit per year.

The average hearing acuity found by Bunch is, even in the age of the fullest human development, i. e., between twenty and thirty years, 10 to 20 SU below the normal accepted as a standard of good hearing by the Western Electric audiometer. Similarly the averages found by Drury are 5 to 10 SU below the normal of the audiometer even in the best hearing groups. Explanation is in the fact that the normal of the audiometric scale was found by tests in a soundproof room, while Drury and Bunch performed their hearing tests in hospital wards. The noise in the average city rooms has a certain masking effect on the ear which, according to Bunch,[16] lowers the air conduction acuity by about 10 Sensation Units.

The averages of Bunch are still further lowered by the choice of subjects consisting of hospital patients. H. C. Montgomery[17] reports tests performed on 200 laboratory employes from twenty to sixty years old, "selected in such a way that the proportion having defective hearing is about what might be found in any normal group of people." His averages do not fall so far below the normal of the audiometer. Montgomery, however, does not report the dispersions of his variations and represents his averages by medians and not by means like Bunch and Drury. (See diagram 11.)

RELATION OF THE AIR CONDUCTION ACUITY TO THE PATHOLOGY OF HEARING.

In spite of the methodical inaccuracies with which the variations of the physiologic acuity have been so far investigated, we have to admit that the range of the variations is very large and that it reaches into the zone of the pathologic possibilities. The level of the air

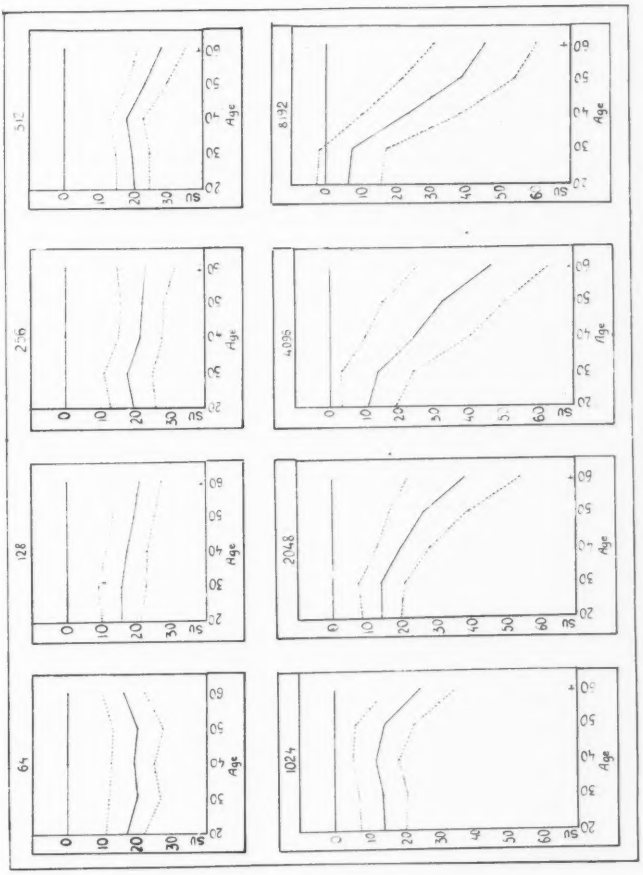


Fig. 10. Age Variations of Air Conduction Acuity According to Bunch.
——— ..... mean variations.

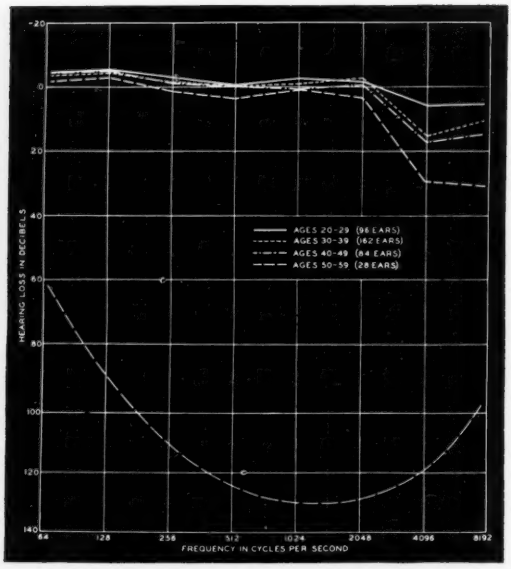


Fig. 11. Age Variations of Air Conduction Acuity According to Montgomery.[12] The curves represent medians.

conduction acuity curve is a definite proof of ear pathology only if the loss of hearing in several frequencies exceeds the lower limit of the physiologic possibilities for the age, ear training, etc., of the person tested.

The air conduction curve may, however, indicate the pathology of the ear not only by its level but also by its shape.

The shape of this curve was studied by several authors. Its precipitous drop was brought into relation with the number of cells in ganglion spirale and with the atrophy of the organ of Corti in the basal part of the cochlea by microscopic examination of several cases performed by Guild.[18] Its drop in low frequencies may be caused by fixation of the stapedius muscle, as was experimentally demonstrated on cats.[19] Dana W. Drury[20] found that luetic infection in early stages is accompanied in about 50 per cent of cases by a sudden and permanent dip of the acuity curve in the frequency 4096.

On the other hand, otosclerosis,[21] toxic influences in pancreatic insufficiency,[22 23] and brain tumors[24] cause changes of hearing acuity without giving the acuity curve a specific shape.

Hardness of hearing caused by acute or chronic inflammatory changes in the middle ear is not characterized by a specific shape of the acuity curve, as has been demonstrated by many authors. In these diseases the shortened hearing of low pitched forks is a real phenomenon, but normal hearing of high-pitched forks is an observational error of the examiner, caused by a short period of vibration of high-pitched forks and by a faulty standardization of the "normal" of the fork against the presbyacoustic ear of the examiner. Audiograms of the several conditions mentioned above are represented in Fig. 12.

The air conduction curve is, as a rule, approximately equally lowered in all frequencies when the conduction apparatus is diseased. This lowering may become compensated to a great extent by obstruction or fixation of the membrane in the round window. This has been experimentally demonstrated by Hughson and Crowe.[19]

It is evident from the above analysis that, by itself, the air conduction acuity curve is only rarely a definite indication of the pathology of the ear. If it were not so and if the level and the shape of the curve were a definite symptom of pathology and of its type, then audiometry would be a biometric method for the determination of the ear pathology. Audiometry is far from that. It is and stays a clinical method, which needs to be supplemented by other tests. It is, however, a method of a much larger precision than the corresponding tuning fork tests.

V. The Bone Conduction Curve.

HEARING ACUITY IN BONE CONDUCTION.

The majority of bone conduction tests are based on two assumptions: 1. By causing acoustic vibration in skull tissues through direct application of the sound source to the surface of the head, such vibrations reach the cochlea without the interference of the conduction apparatus. 2. The proportion of energy transmitted from identical source to the cochlea is the same in each individual.

In the following analysis of bone conduction acuity we shall investigate the truth of the assumptions, and shall interpret the meaning of the bone conduction curve from the standpoints gained through our analysis.

PATHS THROUGH WHICH THE STIMULUS REACHES THE COCHLEA.

The acoustic vibration may reach the cochlea by two different paths: Either by the meatus and through the adjoint conduction apparatus of the middle ear or through the integument and bones of the skull.

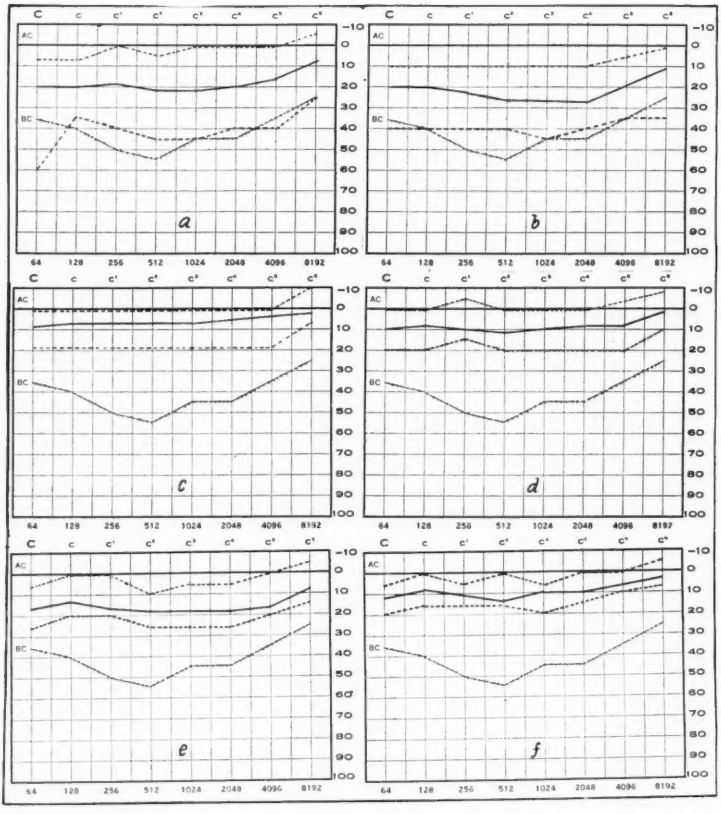


Fig. 12. Different Shapes of Air Conduction Curves.

a. Extensive atrophy in the basal turn of the organ of Corti.[18]
b. Syphilis, seven weeks after infection.[20]
c. Improvement of nerve deafness in pancreatic insufficiency.[23]
d. Temporal tumor.[24]
e. Bilateral acute purulent otitis media.[25]
f. Otosclerosis.[21]
BC - - - - - - normal for bone conduction receiver.

If the sound waves are propagated through the air, and if they carry only a small energy, the hearing in the healthy ear is elicited exclusively by that part of energy which reaches the meatus. Vibrations of the skull, if there are any, are negligible, because their intensity does not reach the threshold of hearing.

However, if the sounding source is in direct contact with the skull tissues, its vibrations are communicated to the cochlear contents by the acoustic waves, which spread through the bones and through the bony capsule of the cochlea.

Tests for bone conduction are by no means "pure" experiments, i. e., the transmission of the vibrations to and by the meatotympanic contents is not and cannot be excluded. Meatus and the middle ear are situated inside a spherical wave which progresses through the head and the meatotympanic contents receive their share of transmitted sound energy.

Consequently by placing the stem of a tuning fork or the end of a bone conduction receiver against the mastoid or other bone of the skull, we test contemporaneously four different paths of sound transmission:

1. *The Cranio-meato-tympanic Path.* — The vibrations spread from the tissues of the head into the air or other contents of the meatus, and from there over the drum membrane and ossicles into the cochlea.

2. *The Cranio-pneumo-tympanic Path.*—The vibrations spread into the air or other contents of the tympanic cavity and from there through the medial tympanic wall into the cochlea.

3. *The Cranio-tympanic Path.* — The vibrations spread from the tissues of the head into the tympanic membrane and into the connected ossicles and are communicated to the cochlea.

4. *The Cranial Path.* — The vibrations spread through the cranial bones and through the osseous capsule of the labyrinth into the cochlea.

Increased or decreased acuity of bone conduction can then be caused either by the changes of the perception apparatus or, at least theoretically, by changes in any of the above paths.

The purpose of the bone conduction tests is to obtain information about the function of the perceiving apparatus without having them modified by the action of the meatotympanic mechanism. The

bone conduction tests will fulfill their purpose only if the influence of the first three (tympanic) transmission paths is negligible or nil or, at least, known.

THE INFLUENCE OF TYMPANITIC COMPONENTS ON BONE CONDUCTION ACUITY OF A HEALTHY EAR.

To appreciate the influence of tympanitic components on bone conduction acuity of a healthy ear would require a following experiment: First, to measure the bone conduction acuity of a healthy ear, then to remove the tympanitic membrane, the malleus and incus, and to again measure the bone conduction acuity after the operation. Such an experiment would be feasible on cats, using the Wever and Bray phenomenon; however, to my knowledge it was not yet performed. To this experiment would correspond a clinical observation, if a case should occur where we had occasion to perform an audiometric measurement of bone conduction of a healthy ear, and again, after a radical mastoidectomy, performed for an intercurrent acute process which did not alter the articulation of the stapes. Such observation is possible, but is not yet recorded in the audiometric literature.

A. G. Pohlman[26] investigated this problem by a statistical method which, though lacking the exactness of an animal experiment, is nevertheless rather convincing. Measurement was performed by the audiometer of Kranz, which continuously increases the frequency on several intensity levels. The principle of his method is statistical comparison of two groups of patients. The intactness of perceiving apparatus is common to both groups. One group, however, has also the conduction apparatus intact, while the tympanitic apparatus of the other group is to such an extent damaged by chronic processes that the air conduction is lowered 30 to 40 Sensation Units. (The first group is Rinné positive, the second is Rinné negative.)

For criterion of intactness of the perceiving apparatus was selected the perception of full extent of frequencies on a certain (for the instrument maximum) intensity level. As the advanced perception defects are usually manifested by the loss of perception of high frequencies, such a criterion is fairly admissible.

The comparison of average acuities of the first group (A, R +) with the second group (A, R-) shows that average hearing through the bone is about the same, no matter whether the conduction mechanism is healthy or damaged. (See diagram 13.)

Considering similar results of Kranz's, Sonntag's and Runge's researches, which will be mentioned later, diagram 13 allows the

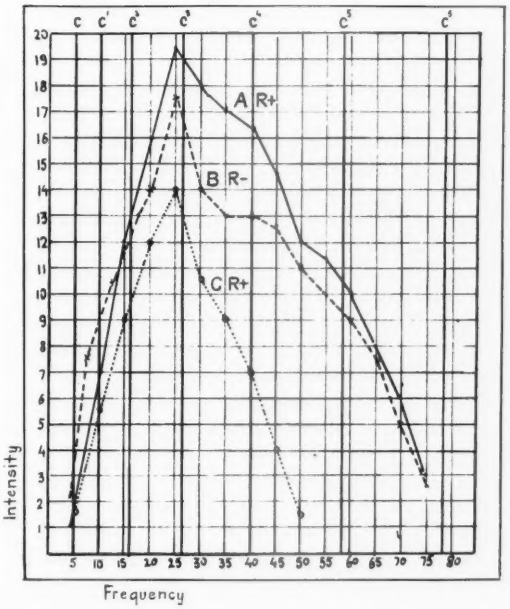


Fig. 13. Influence of the Meato-Tympanical Path on Bone Conduction Acuity According to Pohlman.[26] Audiometer of Kranz. All bone conduction curves.

——— Group *A*, 16 healthy ears, Rinné positive.

x- - - -x Group *B*, 57 Rinné negative ears, with air conduction 30-40 SU lower than that of Group *A*.

o· · · · o Group *C*, 15 Rinné positive ears with perception defect.

Group *A* shows better bone conduction than group *B* only between 126 - 256, because the tests were made in a room, which was not entirely sound-proof. In higher frequencies, Group *A* apparently has a better bone conduction than *B*, because high tones "escaped" from the bone conduction receiver and were heard through the meatus.

following conclusion: In audiometric measuring of bone conduction the tympanitic paths of sound transmission are without practical importance. These paths are unimportant also in the ear which has been damaged by chronic processes to such an extent that the air conduction is lowered by 30 to 40 SU. Consequently the bone conduction acuity is a direct information about the function of the perceiving apparatus irrespective of the function of the transmission mechanism—excepting certain artificial and pathologic changes which shall be mentioned in the following chapters.

Pohlman's study, on which this conclusion is based, has only one methodical defect, namely, that it was performed on a number of cases which is too small for statistical computations (sixteen healthy ears and fifty-seven Rinné negative ears). Statistical method should be supported by a larger number of observations to eliminate the influence of large variations. However, the conclusion is supported by other facts which will be mentioned later.

CHANGES IN THE CONDUCTION APPARATUS WHICH HAVE AN INFLUENCE ON BONE CONDUCTION ACUITY: SCHWABACH'S RULE.

Though we may be convinced that the tympanic apparatus neither lowers nor increases the hearing through the bone, no matter whether we test a healthy ear or an ear affected with chronic tympanic changes, we will have to inquire whether there are any changes in the conduction apparatus which have an influence on the bone conduction acuity.

Such changes exist and are of such character that they can be easily observed, eliminated or allowed for in the interpretation of the bone conduction acuity.

We know from the Gellé test that increased air pressure in an enclosed meatus lowers the bone conduction acuity. We know from the Bing test that closing of the meatus increases the bone conduction acuity. From the Runge test we know that filling of the meatus with water also increases the bone conduction acuity.

Similar pathologic conditions—pus, cerumen, etc.—in the meatus can be easily found before the audiometric examination. The change in the meatal air pressure in Gelle's test is so artificial that it has no equivalent in pathology.

With the exception of Gelle's test we know of no change in the conduction apparatus which would decrease the hearing through the bone. Therefore we could conclude: A decreased bone conduction

acuity is caused exclusively by a lowered function of the perceiving apparatus, assuming that the proportion of energy transmitted from the identical source to the cochlea is equal in all individuals.

This is the fundamental conclusion of Schwabach's rule and it could be used for the interpretation of the bone conduction audiogram if the assumption on which the conclusion is based could be proved true.

The assumption, however, is not true. With equal hearing by air conduction the bone conduction of different individuals varies to a great extent. An example is given in Fig. 14. That can be explained only by an exactly opposite assumption, namely, that the

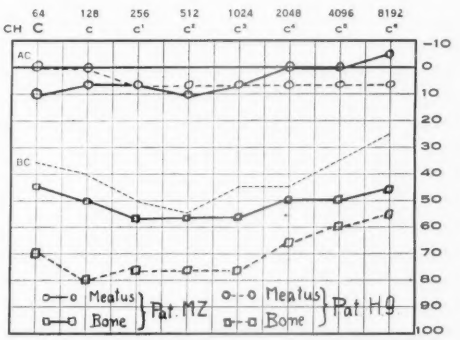


Fig. 14. (Patients, M. Z. and H. G.) Audiograms of two patients with equal air conduction acuities but different bone conductions. History and other examination of both ears are negative. BC - - - normal for bone conduction receiver.

proportion of energy transmitted from identical source to the cochlea is not equal in all individuals.

We see that Schwabach's rule should be expressed with a greater reserve: Decreased bone conduction may be caused by a lowered function of the perceiving apparatus. Such formulation of the rule avoids interpreting the lowered function of the perceiving apparatus as a pathologic phenomenon, because it may be a result of a physiologic variation.

INCREASED BONE CONDUCTION ACUITY.

Increased hearing through the bone is a very well known clinical experience, the explanation and clinical meaning of which had to be modified in many respects by the introduction of audiometry.

The changes of opinions pertaining to the increased bone conduction can be best understood from the analysis of physical and physiologic conditions under which the increased conduction is observed.

In Pohlman's curve (Fig. 13) the bone conduction of Rinné negative average ear is only between 128 to 512 double vibrations, somewhat longer than that of a healthy Rinné positive average ear. Pohlman explains it as a masking of the healthy ear by a minute amount of noise in his examination room. According to Pohlman, the increased bone conduction of the Rinné negative ear does not mean a better hearing through the bone of such an ear but rather a decreased hearing of the healthy average ear caused by the interference of noise which is not perceived by the Rinné negative ear. This question was studied by S. O. Knudsen and J. H. Jones.[27] Their tests were performed by a bone conduction receiver of their own construction on persons with good hearing, both in soundproof and in ordinary rooms. They found that a healthy ear has from 12 to 15 per cent better bone conduction acuity if tested in a soundproof room than when tested in an ordinary room. A control experiment confirmed their finding: If a noise of 30 decibels was introduced into the soundproof room the bone conduction of the healthy ear was lowered 15 per cent. The authors then studied the bone conduction of a pathologic ear in the soundproof and in the ordinary room. They compared the results with a "normal," without, however, stating whether their normal was found in a soundproof or in an ordinary room, or whether they used two normals for comparison. Such a study would have to be undertaken against two normals. The authors do not report whether the masking was more apparent in the low or in the high frequencies. The whole phenomenon would yet deserve a detailed study on ears with definite types of pathology.

The noise in an average city room actually amounts to about 30 decibels,[28] and the new theory is that it decreases the ability of a normal ear to perceive bone vibrations of low intensities. Consequently in the Weber lateralization test the better ear has a decreased bone conduction because it is partially deafened by the noise in the room, while the ear affected with conduction defect has a good or normal bone conduction because it does not perceive the noise. That is why the worse ear lateralizes the tone. The same patient, however, does not lateralize the tone if tested in a soundproof room. The so-called increased bone conduction in such a case is a psychophysiologic phenomenon.

However, the increased bone conduction may result in some cases from a different cause. If we apply a tuning fork to the fore-

head of a person with bilateral normal hearing and occlude one meatus the tone will be lateralized to the occluded side, no matter whether the test is performed in a soundproof or in an ordinary room.[29] In such an experiment one more factor must act different from the meatal deafening of the ear.

Similarly if the meatus is filled with cerumen or with pus, the increase of bone conduction acuity is more than psychologic and occurs even in a soundproof room.[26] The increase evidently takes place through the tympanitic mechanism, because it does not occur if the transmission apparatus of the middle ear is diseased to such an extent that it is unable to transmit sound.[30,31]

If increased bone conduction acuity occurs (under soundproof conditions) only through the action of the conduction mechanism and only after physical changes in the meatus (occlusion or change of contents), then the increase must be effected by the meatotympanic path of the bone transmission. If the fraction of sound energy propagated under ordinary conditions through this path is so small that it adds no loudness to the hearing through the bone, while after the closure of the meatus or after the change of its contents the loudness of the bone conduction increases, then such changes in the meatus must cause energetic changes in the meatotympanic component of the bone conduction. There must be more sound energy delivered from the fork or receiver into the meatal contents. In closing the meatus we probably prevent the escape of sound waves and of their energy from the meatus. The filling of the meatus with liquid or solid material probably improves the transmission of the vibrations from the tissues of the skull into the substance of the filling material. Such energetic changes could be audiometrically measured for different amounts of sound energy, for different frequencies and for different filling materials. Such investigation could have not only theoretical but also practical value—for construction and improvement of hearing aids, which operate on the principle of transmission of amplified sound vibrations through the bone.

Physical prolongation of bone conduction is also effected by an exudate in the middle ear.[26] Pohlman, who explains the changes of the bone conduction caused by fillings as "loading" of the drum membrane, has found that filling of the middle ear cavity is approximately equally effective as filling of the meatus. Prolongation of bone conduction by an exudate in the middle ear could also be explained as increase of the energy transmission through the pneumotympanitic component of the bone conduction.

Bing's test, which is based on the increase of energy in the meatotympanic component of bone conduction, could be worked up audiometrically for different frequencies and different intensities and its diagnostic usefulness could be extended.

INTERPRETATION OF BONE CONDUCTION CURVE.

Masking and energetic changes in the meatotympanic component of bone conduction are important factors for interpretation of bone conduction curves.

Normal threshold of bone conduction is marked in the audiographic blank by a curve which represents an average of an arbitrary group tested in a soundproof room. The consequence of such a choice of normal is that the average of physiologic variations of bone conduction acuities tested in an ordinary room will necessarily fall below the normal of the audiographic blank. It will be so for two reasons: First, because of the masking effect; secondly, because the median of healthy ears belonging to persons of all ages, of all degrees of intelligence, of musical education and of ear training is probably below the average of the group on which the tests of the Bell Telephone Laboratories were performed.

The effect of masking on bone conduction acuity in estimation of E. P. Fowler[32] produces a loss of 10 Sensation Units. Physiologic variations and averages of bone conduction were not yet statistically enumerated. Even if we had statistical data it would be difficult to decide from bone conduction curve alone whether its deviations from "normal" signify pathology or a physiologic variation, unless we knew its variation limits and its specific shapes. Nevertheless, we can state at least general and approximate rules for the interpretation of bone conduction curves found in ordinary rooms:

1. In healthy ears the bone conduction is most frequently somewhat below the "normal," less often coinciding with it and rarely above it.

2. In ears with a chronic obstruction in the middle ear the bone conduction curve coincides with the "normal" of the blank more frequently than in healthy ears. However, the bone conduction of an ear with chronic obstruction may also be higher or lower than the "normal."

3. Ears with closed meatus, or with a meatus filled by a foreign substance and ears with an exudate in the middle ear have their bone conduction curves most frequently above the normal, rarely coincid-

ing or lowered. The effect of filling is not so apparent in high frequencies.[26]

4. Ears with lowered sensibility of the perceiving apparatus have their bone conduction curves lowered. Some of the curves may have a precipitous fall in high frequencies, others may decline gradually.

5. In all ears the hearing through the bone decreases in high frequencies with the progress of age. Severe illness and especially arteriosclerosis causes more rapid progression of loss of high frequencies.[32]

BONE CONDUCTION CURVE AND THE SCHWABACH TEST.

The Schwabach test is an analogy of determination of the bone conduction curve.

The routine Schwabach test, as usually practiced, by comparison of the physician's bone conduction with that of the patient, is actually a comedy full of errors. Patients' bone conduction is declared increased, while in reality the physician's hearing through the bone is decreased by the surrounding noise. An older otologist will not diagnose by it an incipient perception deafness of a young patient, because they both hear the high pitched forks approximately equal number of seconds. In low frequencies, where the perception deafness did not yet reach his ears, the young patient has an "increased" bone conduction—against the physician's presbyacoustic ear. Young otologists with a very good acuity of hearing will often diagnose combined (perception and obstruction) deafness when in reality the process is purely obstructive. Incipient deafness of high tones is not discovered because of a short period of sounding of high pitched forks, etc.

Audiometic studies seem to indicate that the perception factor is a much more frequent element in the etiology of hardness of hearing than heretofore realized. The higher authority of more aged otologists is probably responsible for this chronic mistake.

VI. COMPARISON OF THE BONE CONDUCTION CURVE WITH THE AIR CONDUCTION CURVE.

INTERPRETATION OF AUDIOGRAMS BY COMPARISON OF AIR CONDUCTION AND BONE CONDUCTION CURVES.

E. P. Fowler was the first who offered interpretation of audiograms by comparison of air conduction and bone conduction curves by a rule analogic to the rule of Rinné.[33] Fowler's rule, amplified by

the consideration of physiologic variations and of the effects of masking, could be stated as follows:

1. If all changes in both A.C. and B.C. curves occur in the sense of decreased acuity against the normal of the audiogram, the loss of hearing is probably caused by the perception factor. This factor may not always be of pathologic character and may mean only a physiologic variation if the losses are commensurable with the average acuity for the patient's age, ear training, intelligence, mentality, etc.

2. If the bone conduction curve presents in one or more frequencies a small decrease, normal, or increase above the normal, while the air conduction curve manifests in the same frequency or frequencies a considerable decrease of acuity against the normal of the instrument, the loss of hearing is probably caused by a disturbance in the conduction mechanism of the ear.

3. If in high frequencies both curves show losses, while in low frequencies losses are registered only by the air conduction curve, the loss of hearing is caused probably by an obstructive lesion combined by a perceptive factor.

Such an audiographic diagnostic rule is, like the Rinné rule, only an approximate method of determination of the etiology of deafness, because we are unable to measure the proportion of energy carried by the skull tissues to the cochlea, and because this proportion may be different in each case tested.

To find how great the probability of this rule is would be a complicated biometric problem. Certain limits of this probability could be figured out by an analysis of a large number of cases which have been thoroughly examined clinically and microscopically. And even then the audiographic rule would remain only one of the clinical methods of determination of the pathology of hearing.

INTERPRETATION OF AUDIOGRAMS IN UNILATERAL DEAFNESS.

A more definite interpretation of audiograms is possible if we have an occasion to compare the defective ear with the healthy ear of the same individual.

It is the experience of every otologist who uses an audiometer that both A.C. and B.C. curves of the same individual follow certain similar patterns. This experience is just as frequent in healthy ears as in ears affected with identical pathology. This fact was first reported by Fowler and Wegel.[31]

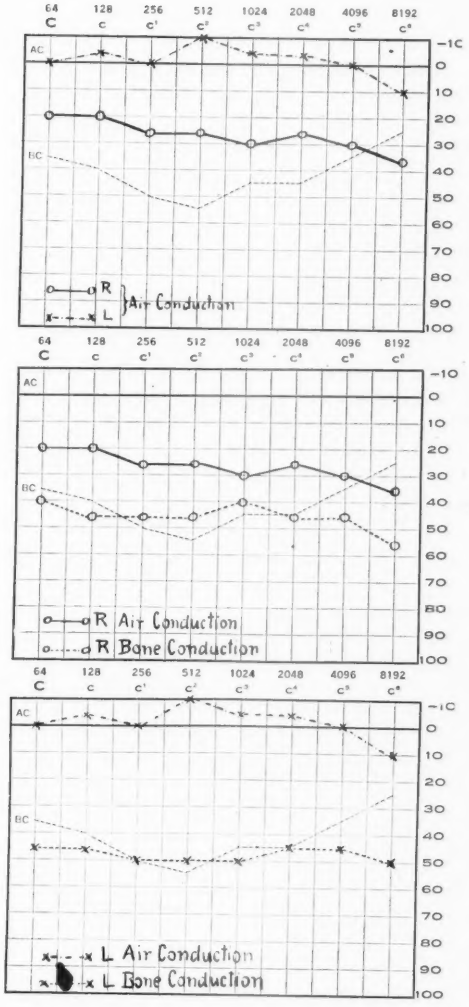


Fig. 15. (Patient, J. B.) Chronic Purulent Otitis Media of the Right Ear. BC - - - normal for bone conduction receiver.

If only one ear is affected, or if the same disease affects one ear more than the other, the audiographic curves can be usually interpreted with a fair amount of precision.

An example is shown in Fig. 15. Right air conduction curve is lower, and at the same time the right bone conduction curve is somewhat higher than the corresponding curves of the left ear. The interpretation of the audiogram is obviously that of an obstructive lesion on the right side. This interpretation is confirmed clinically: The patient was affected with a chronic purulent discharge from the right ear.

Analogically, the lowered level of the bone conduction curve of one ear in comparison with the bone conduction curve of the other ear of the same patient can always be interpreted as lowered function of the perceiving apparatus of the affected ear. At least the author does not know of a case, either from the literature or from his own experience, which would present an audiographic record similar to that of Fig. 14, i. e., equal levels of air conduction curves and different levels of bone conduction curves of the same individual. That leads to a conclusion that the proportions of energy transmitted by an identical source to both cochleæ of the same individual are equal provided we use the same technic and analogic placing of the source while testing either ear.

The considerations contained in this chapter have a certain analogy with the rule of Weber.

VII. Conclusion.

CONCLUSION.

By the use of an instrument of precision for the tests of acuity of hearing, the evasive character of the relations of the hearing acuity to the pathology of the ear has been demonstrated. It cannot be said that the use of the audiometer increases the number of diagnoses in general. Nevertheless, it increases the number of correct diagnoses, supported by definite facts. The medical science cannot be satisfied by diagnostication supported by the authority of the textbooks. And so, though in consequence of audiometic researches, we are in many a case much farther from a "definite" diagnosis than heretofore, nevertheless we are nearer to the discovery of the scientific truth. That is the main contribution of audiometry to otology.

30-28 Thirty-Sixth St.

BIBLIOGRAPHY.

1. Derivations of this and of the previous equations are given in "Speech and Hearing," by Harvey Fletcher. D. Van Nostrand Co., N. Y., p. 308-310, 1929

2. Weber: De pulsu resorptione auditu et tactu annotationes anatomicae et physiologicae, Leipzig, 1934.

3. Fechner: Elem. d. Psychophysic I. 1860.

4. Fletcher, H.: "Speech and Hearing." p. 151, 1929.

5. Hecht, J.: Gen. Phys. Vol. VII, 235, 1929; Vol. XI, 253, 1928.

6. Charpentier: Archive d. Ophthal. Vol. X, p. 108 and following.

7. Fletcher, Harvey: "Speech and Hearing." 1929, p. 158 and following.

8. Fletcher and Munson: "Loudness, Its Definition, Measurement and Calculation." J. Acoust. Soc. of Am., Vol. V, Oct., 1933, pp. 82 and following.

9. Fletcher, Harvey: "Speech and Hearing." p. 140, 1929.

10. Sivian, S. J., and White, S. D.: "On Minimum Audible Sound Fields." J. of Acoust. Soc. of Amer., Vol. IV, pp. 288-321.

11. Ersner, M. S., and Pressmann, J. J.: "An Element of Error in Audiometry." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 40, p. 448, June, 1931.

12. Bunch, C. C.: "Auditory Tests in Unilateral Deafness." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 40, p. 748, Sept., 1931.

13. Guild, Stacy R.: "Correlations of Histologic Observations and the Acuity of Hearing." Acta Otolar., Vol. 17, Fasc. 2-3, p. 244, Fig. 17, 1932.

14. Drury, Dana W.: "Tests of Hearing of Five Hundred Average Ears by the Audiometer 2A." Arch. of Otolaryngology, Vol. 1, p. 524, May, 1925.

15. Bunch, C. C.: "Age Variations in Auditory Acuity." Archives of Otolar., Vol. 9, p. 624, June, 1929.

16. Bunch, C. C.: "The Use of Audiometers in Clinical Work." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 41, p. 714, Fig. 1, A-B, Sept., 1932.

17. Montgomery, H. C.: Bell Lab. Record, pp. 311-313, May, 1932.

18. Guild, Stacy R., Crowe, S. J., Bunch, C. C., and Polvogt, L. M.: "Correlations of Differences in the Density of Innervation of the Organ of Corti with Differences in the Acuity of Hearing, Including Evidence as to the Location in the Human Cochlea of the Receptors for Certain Tones." Acta Otolar., Vol. 15, Fasc. 2-3, pp. 269-308, 1931.

19. Hughson, Walter, and Crowe, S. J. Acta Otol., Vol. 18, Fasc. 3, p. 291.

20. Drury, Dana W.: "Deafness in Syphilis." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 38, p. 651, Sept. 1929.

21. Bunch, C. C.: "Some Observations on Clinical Otosclerosis." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 43, p. 344, June, 1934.

22. Drury, Dana W.: "An Aural Syndrome of Hepatic Origin." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 40, p. 415, June, 1931.

23. Gottlieb, M. J.: "Deafness, Hepatic Dysfunction, Pancreatic Insufficiency: A Clinical Entity." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 41, p. 523, June, 1932.

24. Drury, D. W.: "Aural Acuity and Brain Lesions." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 40, p. 682, Sept., 1931.

26. Bunch, C. C.: "Auditory Acuity Tests." Arch. of Otolaryngology, Vol. 3, p. 114.

26. Pohlman, A. G.: "Correlations Between the Acuity for Hearing Air and Bone Transmitted Sounds in Rinné Negative and Rinné Positive Cases." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 39, p. 927, Dec., 1930.

27. Knudsen, V. O., and Jones, J. H.: "Bone Conduction." Archives of Otolaryngology, Vol. 13, p. 503, April, 1931.

28. Fletcher, Harvey: "Advice to the Hard of Hearing." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 41, p. 726, Sept., 1932.

29. Hallpike: "Some Observations on Bone Conduction." J. of Lar. and Otol., Vol. 45, p. 14, Dec., 1930.

30. Pohlman and Kranz: "Binaural Acuity for Air and Bone Transmitted Sound." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Dec., 1925.

31. Runge: Zeitschrift fur Hals, Nasen und Ohrenheilkunde, Vol. 5, p. 289, 1923.

32. Fowler, E. P.: "Effect of Severe Illness Upon the Hearing." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 43, p. 400, June, 1934.

33. Fowler, E. P.: "Limitations of the Functional Tests in the Differential Diagnosis of Deafness." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 38, p. 766, Sept., 1929.

34. Fowler, E. P., and Wegel, R. L.: "Audiometric Methods and Their Applications." Transactions of the Triological Soc., p. 98, 1922.

Joseph W. Bryan

JOSEPH HAMMOND BRYAN.

1856-1935.

Dr. Joseph Hammond Bryan, the son of Joseph Brook and Louisa Stearns (Hammond) Bryan, was born in Washington, D. C., July 4, 1856.

After a preliminary education in the schools of his native city, he went to the University of Virginia, with the intention of taking a course to become an engineer; but later changed his mind, and took up medicine. In 1877 he earned there the degree of M. D.

Following his graduation he spent a year as interne in Charity Hospital, Blackwell's Island.

In 1880 he entered the Navy as Assistant Surgeon, and remained in the service until 1885, when he resigned to take up the specialty of otolaryngology.

He studied two years in European medical centers, returning to begin the practice of the specialty in his native city in 1887. He was one of the first three specialists in this branch in Washington, the other two having been Dr. Carroll Morgan and Dr. Frank Hyatt, these two having likewise prepared themselves by extensive study abroad. Dr. Bryan quickly rose into prominence and obtained a large and lucrative practice.

From the beginning he took an interest in the scientific aspects of his profession. His first contribution, in 1888, was on the important subject of cerebellar abscess complicating ear disease.

From year to year he continued to write articles of value that generally displayed originality of thought, and did much to advance in those early days the progress of his specialty. He was one of the first to recognize the important rôle of the sinuses and to advocate and devise rational methods of treating them.

He wrote as early as 1889 a paper on suppurative diseases of the antrum, in 1893 on suppurative ethmoiditis and in 1897 he made a noteworthy contribution to the surgery of frontal sinus diseases, advocating the necessity of providing nasal drainage in connection with external opening through the anterior wall.

Dr. Bryan was also a pioneer in the establishment of the relationship between sinus disease and the eyes, reporting from time to time cases substantiating this idea.

In the beginning of the war, Dr. Bryan volunteered his services and was appointed, June 18, 1917, Major, Medical Corps of the Army. After the war he remained in the reserves as Colonel.

He was one of the founders of the Episcopal Eye, Ear and Throat Hospital of Washington, and he remained actively on the staff to the end of his life. He was a member of the American Laryngological Society, and for a number of years served as its librarian. He always took an active part in its proceedings.

Dr. Bryan was a man of keen intellect, sound judgment and sturdy character. He was positive in his opinions and fearless in the expression of them.

PUBLICATIONS OF DR. JOSEPH H. BRYAN.

1. Cerebellar Abscess Due to Diseases of the Ear. J. Am. Med. Assoc., Chicago, X, 176, 1888.

2. Diagnosis and Treatment of Abscess of the Antrum. J. Am. Med. Assoc., XIII, 1889.

3. Some Recent Advances in the Diagnosis and Treatment of Abscess of the Antrum. Virg. Med. Month., Richmond, XVII, 18-30, 1890-91.

4. Acute Edema of the Larynx with the Report of a Case Resulting from Pyemia. Med. News, Phila., LX, 1892.

5. A Case of Suppurating Ethmoiditis. N. Y. Med. J., LVII, 97, 1893.

6. Diseases of the Accessory Sinuses of the Nose. Syst. of Dis. of Ear, Nose and Throat, 1, 743, 1893.

7. Surgery of the Maxillary Sinus. Tr. Cong. Am. Phys. and Surg., New Haven, 111, 217, 1894.

8. The Surgical Treatment of Chronic Suppurating Otitis Media. J. Am. Med. Assoc , Chicago, XXV, 700-704, 1895.

9. A Further Contribution to the Study of Suppurative Diseases of the Accessory Sinuses with Report of Cases. N. Y. Med. J., LXII, 450-456, 1895.

10. A Contribution to the Study of Suppurative Diseases of the Accessory Sinuses with Report of Cases. Med. Rep., London, VII, 81-85, 1896.

11. A Contribution to the Study of the Anatomy of the Fronto-ethmoidal Region. Brit. Med. J., London, II, 1393, 1897.

12. Catarrhal Inflammation of the Upper Air Passages as They Appear in Washington and Vicinity. Nat. Med. Review, Wash., VII, 182, 1897-98.

13. On the Treatment of Chronic Frontal Sinusitis by Means of an Opening Through the Anterior Walls of the Sinus and Drainage Through the Nose. N. Y. Med. J., LXVI, 451-4, 1897.

14. Treatment of Empyema of the Frontal Sinus. J. Am. Med. Assoc., Chicago, XXX, 483, 1898.

15. Report of Two Cases of Suppurating Mastoiditis. J. Am. Med. Assoc., Chicago, XXX, 534, 1898.

16. Further Results of Operative Treatment of Chronic Frontal Sinusitis. N. Y. Med. J., LXVIII, 881, 1898.

17. On the Relation of Disease of the Accessory Cavities to Diseases of the Eye. J. Am. Med. Assoc., Chicago, Nov., 1899.

18. Pemphigus Chronicus Vulgaris of the Larynx and Mouth. N.Y. Med. J., 1899.

19. Chronic Abscess of the Frontal, Ethmoidal and Sphenoidal Sinuses, Followed by Meningitis and Death. N. Y. Med. J., Jan., 1900.

20. A New Aural Syringe. N. Y. Med. J., LXXIII, 263, 1901.

21. Suppurating Mastoiditis with Report of Cases; Suppurating Otitis Media, Both Ears; Suppurating Mastoiditis on Right Side, Abscess Extending into Deeper Tissues of Neck and Extradural Abscess. J. Am. Med. Assoc., Chicago, XXXVI, 1311, 1901.

22. Chronic Empyema of the Frontal, Ethmoidal and Sphenoidal Sinuses, with Extensive Necrosis Complicated with Adenoma of the Posterior Ethmoidal and Sphenoidal Region. Am. J. Med. Sc., Philadelphia, CXXIV, 416, 1902.

23. Malignant Diseases of the Tongue with a Report of Two Cases. Tr. Am. Laryng. Assoc., N. Y., 19-23, 1906.

24. Report of a Case of Removal of Foreign Body, a Grain of Corn, from the Trachea of a Child Six Years Old. Tr. Am. Laryng. Assoc., N.Y., XXX, 99-101, 1908.

25. Operative Treatment of Suppurative Sinus Diseases, Producing Orbital Complications. J. Am. Med. Assoc., Chicago, LVII, 624, 1911.

26. A Further Contribution to the Study of Diseases of the Accessory Sinuses in Relation to Diseases of the Eye and the Surgical Methods to Be Adopted for Their Relief. Surg., Gynec. and Obst., Chicago, XIV, 565, 1912.

27. On the Relation of Diseases of the Posterior Sinus to Painful Conditions of the Ear. Tr. Am. Otol. Soc., New Bedford, Mass., XII, pt. 3, 481, 1912.

28. Report of a Case of Myxofibroma of the Nasopharynx. Tr. Am. Laryng. Assoc., N. Y., XXXIV, 302, 1912.

29. Report of a Case of Ulceration of the Larynx; Perichondritis of the Arytenoid Cartilages, Abscess and Partial Exfoliation of Both Cartilages Resulting from Typhoid Fever. Annals of Otology, Rhinology and Laryngology, St. Louis, XXII, 811, 1913.

30. Report of Two Cases of Mastoid Abscess, Infective Thrombosis of the Latent Sinus and Jugular Vein; Resection of the Jugular; Recovery. Tr. Am. Otol. Soc., New Bedford, XIII, pt. 3, 424, 1915.

Ibid., Annals of Otology, Rhinology and Laryngology, St. Louis, XXIV, 543, 1915.

31. On the Relation of Diseases of the Accessory Sinuses to Diseases of the Eyes, Especially in Children; with a Report, Two Cases. J. Laryng., London, XXXII, 68, 1917.

32. Nichols and Bryan: The Tonsils as Foci of Infection in Streptococcus Hemolyticus Carriers. J. Am. Med. Assoc., Chicago, LXXI, 1813, 1918.

33. A Symposium on Streptococcus Hemolyticus Carriers: Clinical Aspect. Annals of Otology, Rhinology and Laryngology, St. Louis, XXIX, 337, 1919.

34. Report of a Case of Otits Media Suppurative, Chronic, Right, Mastoiditis Operation Followed by Meningitis and on Postmortem Examination Findings of Old Abscess in the Temporosphenoidal Lobe of the Brain. Tr. Am. Otol. Soc., New Bedford, Mass., XV, 93, 1919.

Ibid.: Annals of Otology, Rhinology and Laryngology, St. Louis, 1083, XXXVIII, 1919.

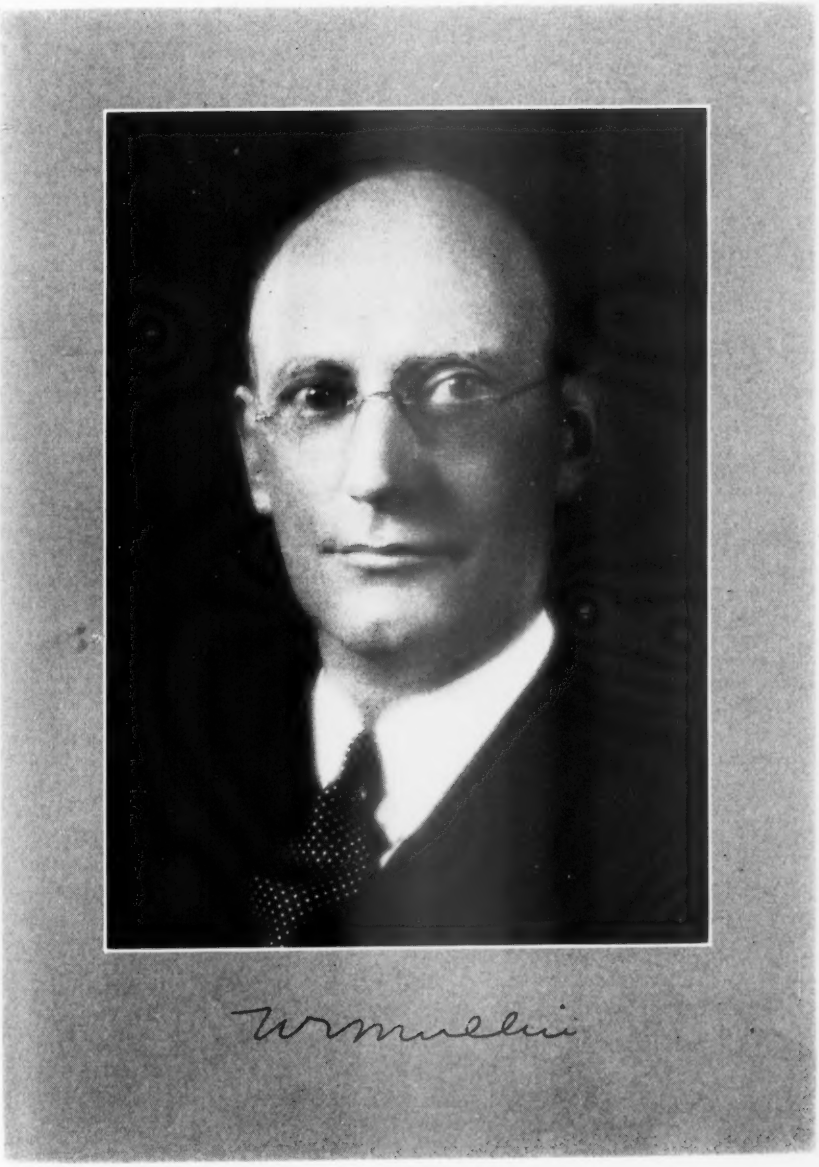
35. Report of a Case of Influenza Followed by Pneumonia, Laryngitis, Otitis Media Suppurative, Acute, Right Side; Tonsillitis Hypertrophic, Chronic Bilateral, Streptococcus Type. Annals of Otology, Rhinology and Laryngology, St. Louis, 1009, XXXVIII, 1919.

36. Report of a Case of Extradural Abscess and Subdural Abscess Following Suppurative Frontal Sinusitis and Osteomyelitis of the Frontal Bone. Am. J. Med. Sc., Philadelphia, 687, CLX, 1920.

37. Suppuration of the Lungs: Laryngologic Aspect. Arch. Otolaryng., Feb., 1928.

38. Abscess of the Brain of Otitic Origin. Annals of Otology, Rhinology and Laryngology, XXXVII, Sept., 1928.

39. Report of a Case of Papillary Carcinoma of Maxillary Sinus. Annals of Otology, Rhinology and Laryngology, XLI, Dec., 1932.



WILLIAM V. MULLIN.

1884-1935.

Dr. William V. Mullin of Cleveland died on April 24th, of septicemia, following a very short illness.

He was born in Iowa City, Iowa, February 14, 1884. He attended the State University of Iowa and then went to Colorado and finished his medical studies at Denver and the Gross Medical College of the University of Denver, and graduated in 1908. He interned in St. Joseph's Hospital in Denver, and when he had completed his internship he started his general practice in southern Colorado. In 1912 he took up the specialty of Ear, Nose and Throat in Colorado Springs, Colorado. Following this he took his post graduate work in Otolaryngology in Berlin in 1914. In 1926 he moved from Colorado Springs to Cleveland, Ohio, where he became associated with the Cleveland Clinic as head of the Department of Otolaryngology.

He was the former Vice-President and Treasurer and present Secretary of the American Laryngological Association. He was also past Secretary and Chairman of the Section of Laryngology, Rhinology and Otology of the American Medical Association, and Secretary for Instruction of the American Academy of Ophthalmology and Otolaryngology. He was a member of the American Board of Examiners in Otolaryngology and of the Editorial Board of the Annals of Otology, Rhinology and Laryngology, and the author of many articles on otolaryngology.

Dr. Mullin was a member of the Cleveland Academy of Medicine; the Ohio State Medical Association; fellow of the American Medical Association and of the American College of Surgeons. He was a member of the American Academy of Ophthalmology and Otolaryngology and also a member of the American Laryngological, Rhinological and Otological Society, the American Otological Society and American Laryngological Association.

Through his untimely death American Otolaryngology loses one of its most active exponents. His interest and scientific curiosity, his passion for unvarnished facts, will insure for his contributions a permanent place in the literature of the specialty. His investigation of the lymphatic drainage of the nasal sinuses with C. T. Ryder, published in March, 1921, was widely quoted.

But most of all he will be mourned as a true friend whose enthusiasms contributed much to the Societies in whose activities he took such a prominent part. He will live long in the affectionate memory of his fellows.

PUBLICATIONS OF DR. WILLIAM V. MULLIN.

1. Abscess of the Left Frontal Lobe Following Suppuration of the Frontal Sinus: Report of a Case and Exhibition of Specimen. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 27:667, June, 1918.

2. The Lymph Drainage of the Accessory Nasal Sinuses. Trans. Am. Laryngol., Rhinol. and Otol. Soc., page 73-103, 1919.

3. Indifference of the Laryngologist Toward Tuberculous Laryngitis and the Tuberculosis Problem. J. A. M. A., 75:300-301, 1930.

4. Experimental Lesions of the Lungs Produced by the Inhalation of Fluids from the Nose and Throat. Am. Rev. Tuberc., 4:683-687, November, 1930. (With C. T. Ryder.)

5. Studies on Lymph Drainage of Accessory Nasal Sinuses. Laryngoscope, 31:158, March, 1921. (With C. T. Ryder.)

6. Deformity of Nasal Bones Following Chronic Nasal Suppuration in Rabbit. Laryngoscope, 31:634, August, 1921.

7. Accessory Sinuses as Etiologic Factor in Bronchiectasis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 30:683, September, 1921.

8. Value of Direct Inspection in the Diagnosis of Chronic Maxillary Sinus Disease. Laryngoscope, 32:300-303, April, 1922. (With F. L. Dennis.)

9. The Management of Tuberculous Laryngitis. Southern M. J., 15:501-506, June, 1922.

10. Pollen and Hay Fever: A Regional Problem. Trans. Am. Acad. of Ophth. and Otolaryng., page 467-477, 1922; also, Colorado Med., 20:96-100, April, 1923.

11. An Analysis of Some Cases of Tubercles in the Tonsil. J. A. M. A., 80: 1211-1213, April 28, 1923.

12. Diagnostic Limitations in Laryngology. Trans. Am. Laryngl. Assn., 46:207, 1924; also, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 34:197-202, March, 1925.

13. The Relation of Paranasal Sinus Infection to Disease of the Lower Respiratory Tract. J. A. M. A., 87:739-741, Sept. 4, 1926.

14. Rhinological Aspect of Bronchial Asthma. Radiology, 8:482-487, June, 1927.

15. The Routine of the Specialist and His Relations to Other Branches of Medicine in the Diagnosis of Ear, Nose and Throat Disease. Memphis Med. J., July, 1927.

16. Studies of Pathologic Tissue Removed from Chronically Infected Nasal and Accessory Sinuses: A Preliminary Report. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 37:182-195, March, 1928. (With R. P. Ball.)

17. The Physiology of the Larynx. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 37:627-630, June 1928. Also: Trans. Am. Laryng., Rhin. and Otol. Soc., 33:505-508, 1927.

18. What Is the Relation of the Thyroid to the Larynx? Trans. Am. Laryng. Assn., 50:30-35, 1928.

19. Accessory Sinus Disease as It Concerns the General Practitioner. Kansas City Southwest Clinical Bulletin, 1929.

20. The Anatomy, Physiology and Diseases of the Salivary Glands. Trans. Am. Acad. Ophth. and Otolaryngol., 34:354-363, 1929.

21. The Work of the Section on Laryngology, Otology and Rhinology, Chairman's Address. Sec. L., R. and O. of A. M. A., J. A. M. A., 94:312-313, February 1, 1930.

22. Malignant Tumor of the Breast with Metastasis to the Opposite Side of the Larynx and Contralateral Vocal Cord Paralysis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 39:125-127, March, 1930.

23. The Management of Chronic Sinus Disease. New York St. J. Med., 30:1280-1282, Nov. 1, 1930.

24. Symposium on the Relation of Sinus Disease to Pulmonary Infection from the Standpoint of the Rhinologist. Trans. Coll. Physicians of Philadelphia, 52:245-247, 1930.

25. The Salivary Glands: 1930 Proceedings of the Interstate Post-Graduate Medical Assembly of North America, Minneapolis (1931), pages 91-94.

26. Experience with Chronic Deafness. Illinois M. J., 60:70-73, July, 1931.

27. The Filament-Nonfilament Count: Its Diagnostic and Prognostic Value. J. A. M. A., 97:1133-1138, Oct. 17, 1931.

28. Diagnosis and Treatment of Cancer of the Larynx. Proc. Interstate Post-Graduate Medical Assembly of North America (1931), 198-200, 1932.

29. The Interrelation of Allergy and Otolaryngology. Arch. Otolaryng., 15: 413-417, March, 1932.

30. Allergy. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:387-392, June, 1932.

31. The Relation of the Thyroid Gland to Disturbance of the Larynx: The Nose, Throat and Ear and Their Diseases; Jackson-Coates, pp. 802-803. W. B. Saunders & Co.

32. Tumors of the Mouth, Pharynx and Fauces: The Nose, Throat and Ear and Their Diseases; Jackson-Coates, pp. 322-331. W. B. Saunders & Co.

33. Laryngeal Disturbances in Hyperthyroidism: Diagnosis and Treatment of Diseases of the Thyroid Gland; George Crile and Associates; pp. 176-180. W. B. Saunders & Co.

34. Review of Sinus-Chest Infections. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:794-804, Sept., 1932. Also Trans. Am. Laryng. Assn., 54:48, 1932.

35. The Diagnosis and Treatment of Cancer of the Larynx. J. Iowa St. Med. Soc., 22:525-529, Nov., 1932.

36. Circumscribed Arachnoid Cyst Giving Symptoms of an Acoustic Neuroma. Laryngoscope, 43:213-229, March, 1933.

37. Principles of Modern Surgery in Otolaryngology. Trans. Am. Acad. Ophth. and Otol., 1933.

38. The Present Status of Infection of the Upper Respiratory Tract in Its Relation to Focal Infection. The New England J. Med., p. 50, Jan. 10, 1935.

NOTICE.

III. Internat. Oto-Rhino-Laryngologen-Kongress.

At the International Oto-Rhino-Laryngological Congress held in Madrid in 1932, a resolution was passed to accept the invitation of the "Gesellschaft Deutscher Hals- Nasen- und Ohrenärzte" and the "Otolaryngologische Gesellschaft zu Berlin," and to arrange the next meeting in the summer of 1936 in Berlin.

Accordingly the dates for the next meeting have been set for Monday, August 17th to Saturday, August 22nd, directly following the Olympic Games.

The following items have been selected for the program:

1. *Radiotherapy of Malignant Tumors of the Nose and Throat.*
   Discussions by:
   - Dr. E. Berven, Stockholm
   - Prof. H. Coutard, Paris
   - Prof. Hautant, Paris
   - Prof. J. Maisin, Louvain
   - Prof. W. Palumbo, Florence
   - Prof. F. Perusia, Milan
   - Prof. Cl. Regaud, Paris
   - Prof. J. L. Roux-Berger, Paris
   - Prof. A. Torrigiani, Florence

2. *Electro-auditory Apparatus for Aids to Hearing and for Examination of Hearing.*
   Discussions by:
   - Priv. Doz. Dr. Langenbeck, Leipsic
   - Prof. Erwin Meyer, Heinrich Herz-Institut, Berlin

3. *The Influence of Constitutional Disorders Upon the Ear, Nose and Throat.*
   Discussions by:
   - Prof. Albrecht, Tübingen
   - Prof. van Gilse, Utrecht

The names of other participants not yet selected and details of the program together with that of the social functions and an account of the facilities for traveling and accommodation will be sent later with the official invitation.

v. Eicken, President.

Berlin, Lutzowplatz 7.

Abstracts of Current Articles.

NOSE

Morphology of the Lamina Cribrosa of the Ethmoid (Sulla morfologia della lamina cribrosa dell'ethmoide.)

Ciurlo, L. (Milano), Arch. Ital. di Otol., Rino. and Laring., 6:419-446 (June), 1934.

A strictly anatomic but interesting article containing nineteen photographs and four large classification tables.

The author studied the cribriform plate of 500 adult skulls (300 male and 200 female), classifying the various forms in the order of their frequency as follows: 1. Cuneiform and trapezoid, 40 per cent; 2. Rectangular, 30.2 per cent; 3. Ogival and mitral, 12.2 per cent; 4. Rhomboid and ellipsoid, 11.2 per cent; 5. Very few irregular.

Then he discusses the hypothesis of Forster and Augier on the genesis of such diversities, but he believes that the shape of the lamina cribrosa is influenced by the size of anterior cerebral lobes, sinuses and constitutional or hereditary characteristics. Therefore, he divides these skulls according to their cephalic, facial and nasal indexes, in order to determine the relation between the various forms of lamina cribrosa and certain types of crania.

Ciurlo's final conclusions are that the cephalic measurements do not specify any particular type of lamina cribrosa; the facial index is frequently found correct; while the nasal index is the most reliable in determining a definite form of cribriform plate.

The author considers this last index as being the most logical, because the lamina cribrosa and the nose have common morphnelogic, physiologic and anatomic relations.

SCIARRETTA.

Malignant Tumors of the Nasal Mucosa.

Price, L. Woodhouse (Glasgow), J. of Laryng. and Otol., 50:153 (Mar.), 1935.

The author reviews a series of cases of malignant tumors arising in the mucosa of nose and nasal accessory sinuses, and anatomic reasons are advanced for the highly lethal properties of these tumors. The difficulty of clinical diagnosis of nasal tumors is discussed and a

plea for early cytologic examination of aspirated fluid is made. The pathology of nasal tumors is given and a simple classification suggested according to the nature of the epithelium in which they arise.

The invasion of the nasal fossæ and nasal accessory sinuses by tumors of extra-nasal origin is discussed. The complexity of tumors arising from paradental epithelium is shown to be attributable to their origin from specific or multipotential cells derived from "anlage" of the enamel organ.

GOLDSMITH-MCLEOD.

Influence of the Accessory Sinuses on Breathing (Nasen nebenhöhlen und Atemmechanismus).

Kerekes, G. (Budapest), Acta Otolar., 21:4-438, 1935.

Using manometric tracings from cannulas tightly inserted into the maxillary antra of humans and the frontals of dogs, Kerekes has decided that given equal conditions of barometric and other extrinsic pressure, the slight fluctuations of air pressure within the sinuses during breathing are of great physiologic importance.

He considers the sinus cavities to be organs of accommodation, which assist in fitting our breathing movements to stimuli furnished by the air outside. Interruption to this free flow through tissue changes, edema, fluid or exudate has a reflex action which may not only speed up the breathing rate, but may give rise to minus-pressure headaches or neuralgias, even if no visible inflammation be present.

FENTON.

Rhinoscleroma: Report of Two Cases.

Schwartz, Alfred A. (New York), Arch. of Otolaryng., 21:199 (Feb.), 1935.

Two cases of advanced rhinoscleroma with nasal, laryngeal and tracheal involvement are reported.

In both cases improvement followed roentgen therapy. Neither roentgen nor radium therapy should be considered a cure for scleroma. These patients must be kept under continual observation and treatment with roentgen rays must be repeated when necessary. Treatment with roentgen rays appears to be more efficacious than treatment with radium.

Except in case of urgent necessity or for securing material for biopsy, surgical intervention is contraindicated in cases of scleroma.

TOBEY.

PHARYNX

Retropharyngeal Tumors (Des tumeurs rétropharyngées).

Natanson, L. (Moscow), Rev. de Lar., Otol., Rhin., 56:51 (Jan.), 1935.

This is a review of eighty-five collected cases of the past seventy-five years, with report of three new ones. Most of them are neurofibromas from the cervical sympathetic; next come lipomata. Accessory thyroids are rare. The Horner-Bernard syndrome assists with the diagnosis. Operation should be made before the growth has extended too high behind the soft palate. Small growths (not thyroid) are best removed through the mouth; all large infiltrating growths, by lateral pharyngotomy.

Since these growths are usually benign, radium or the roentgen ray only complicates their eventual removal. Frankly malignant growths of course demand radiation. Such growths in babies are always malignant.

FENTON.

LARYNX

Diagnosis and Surgical Treatment of Tuberculosis of the Larynx.

Schugt, Henry P. (New York), Arch. of Otolaryng., 21:175 (Feb.), 1935.

Direct laryngoscopy for a correct diagnosis is preferable to indirect examination with a mirror. Emphasis is laid on the importance of the laryngeal ventricle in incipient cases of laryngeal tuberculosis.

Electrocautery is of great value in the routine treatment of the tuberculous larynx. The curative paralysis in one-sided involvement is a valuable method, although little is known of it. In cases of laryngeal obstruction from tuberculous lesions tracheotomy should be avoided and replaced by direct intralaryngeal operations. In treating dysphagia (pain in swallowing) blocking of the superior laryngeal nerve is the method of choice. Ordinarily this is done from the outside. The nerve can also be blocked from the inside through the pyriform sinus. Resection of the nerve should be limited to patients in good general condition. In cases of pharyngeal tuberculosis severe pain can be dealt with by blocking the pharyngeal nerve plexus. Small lesions in the mouth can be excised and treated locally with success. In advanced cases of lingual tuberculosis with severe pain, conservative treatment may be useless. The pain in this area can be destroyed either by blocking the lingual nerve with alcohol intraorally or by resecting the nerve from the outside.

TOBEY.

Cancer of the Larynx: Observations in Two Hundred Consecutive Cases.

Tucker, Gabriel (Philadelphia), Arch. of Otolaryng., 21:1 (Jan.), 1935.

The study confirms the already well established fact that the early symptoms of cancer of the larynx are two in number: (a) Hoarseness and (b) local discomfort. One or both of these symptoms occurred in every case in this series while the disease was in an early stage. Vocal abuse or the excessive use of tobacco were probably predisposing causes in more than 50 per cent of the cases.

The following instances of complicating and concurrent pathologic conditions were noted: Cancer of the larynx with tuberculosis of the larynx and lungs, one case; cancer of the larynx with tuberculosis of the lungs, eight cases; cancer of the larynx with evidence of syphilitic infection, seven cases; cancer of the larynx, pulmonary tuberculosis, constitutional syphilis and diabetes, one case; cancer of the larynx (intrinsic) and cancer of the esophagus (middle third), one case (on histologic examination basal cell cancer with a different type of cell at each location was found) and cancer of the larynx, pulsion diverticulum of the esophagus and constitutional syphilis, one case.

Papillomas, keratosis, leucoplakia and chronic localized inflammatory diseases are noted as precancerous condition.

The study confirms the belief that a large proportion of all cases of cancer of the larynx are intrinsic in origin. In this series 144 cases of the 200 investigated, or 72 per cent, were probably of intrinsic origin.

TOBEY.

Malignant Disease of the Larynx and Pharynx.

Stewart-Harrison, R., and Sarasin, R. (Zurich), J. of Laryng. and Otol., 50:233 (Apr.), 1935.

Two previous papers have been published by one of the writers which dealt chiefly with the results of radiation therapy in malignant disease of the larynx and pharynx at Zurich. In this article the technic of treatment as practiced at the present time is described.

The reactions of the various tissues are discussed. The skin reaction is not a limiting factor, nor is it an indicator for treatment. It is reduced or prevented by the application of red or infra-red rays. The reactions of the blood, salivary glands, blood vessels, nerves and muscles are but rarely decisive factors in the treatment. The reaction of the mucous membrane is important as an indicator and, in view of the effect of this reaction on the general condition, the reaction of

the substrate tissues is of supreme importance. Damage to the substrate, either by the tumor or by the treatment, leads to a reduction in the relative sensitivity of the tumor. Infiltrating and infected tumors, recurrences and incompletely treated tumors begin with a damaged substrate and are resistant to treatment. The technic must be modified in accordance with the relative sensitivity of each individual case. The more resistant a tumor the slower is its reaction to radiation and the longer must treatment be continued. The time factor may vary from 20 to 100 days. The size of each individual dose and the total dose are dependent upon this time factor. The time factor must be chosen to avoid the possibility of further damage to the substrate and a further reduction in the relative sensitivity of the tumor. It will therefore be subject to modifications during the course of treatment, from day to day, according to the reactions, local and general, presented by the patient. A long time factor and large fields demand slight local reactions, therefore small individual doses. At the same time the intensity of the radioepithelitis is reduced, preserving the general condition. Only when the treatment is exactly suited to the substrate and the tumor will this form of treatment be relatively safe and attended by encouraging results.

GOLDSMITH-MCLEOD.

"Dysphonia Plicae Ventricularis." Phonation with the Ventricular Bands.

Jackson, Chevalier, and Jackson, Chevalier L. (Philadelphia), Arch. of Otolaryng., 21:156 (Feb.), 1935.

Phonation with the ventricular bands is a rather frequent and usually unrecognized cause of hoarseness.

It may be a vicarious dysfunction with a pathologic basis or a physiologic compensation for lost or impaired vocal cords.

When it is a desirable compensatory function a good voice can be developed by systematic training.

When it is a usurpation of function by aggressively overactive ventricular bands, or when the resumption of activity by temporarily disabled vocal cords renders persistent phonation with the ventricular bands undesirable, this phonation can be checked by training, or if necessary, by operation.

The operation consists in nipping out a bit of tissue from the middle of the free edge of one or both ventricular bands. Before this is done, however, it is necessary to be certain that the vocal cords can approximate, draw tense and vibrate.

TOBEY.

Indications and Results of Surgical Treatment of Cancer of the Larynx (Des indications et des résultats du traitement chirurgical du cancer larynx).

Portmann, G., and Despons, J. (Bordeaux), Rev. de Lar., Otol., Rhin., 56:5 (Jan.), 1935.

Professor Portmann throws the weight of his experience against roentgen or radium therapy for cancer of the larynx, except as a late palliative. He favors, instead of cordectomy, lateral hemilaryngectomy, or resection of the anterior half of the thyroid cartilage for various locations of intrinsic cancer of limited extent. But diffuse cancer requires total laryngectomy without delay.

Commenting on the need of early diagnosis, Portmann reports on forty-six endolaryngeal cancers during eight years, in which period 200 laryngopharyngeal cancers were also studied. This monograph of fifty pages deserves careful and detailed reading.

FENTON.

Paralytic Stenosis of the Larynx Treated by Laryngostomy (Stenosi paralitiche della laringe trattate con la laryngostomia).

Gibelli, S. L. (Genova), Valsalva, 10:493-500 (July), 1934.

Gibelli reports two cases of laryngeal stenosis produced by a bilateral paralysis of the crico-arytenoideus posterior.

He discusses the mechanism which brings about approximation of the cords, also the various technics employed for the correction of this fatal condition.

The author finally concludes by describing the following technic, which he considers new, as far as his bibliographic searches have gone.

An extensive laryngotomy is done. The incision will involve a good portion of the thyrohyoid membrane and the first rings of the trachea, thus permitting displacement and dislocation of the thyroid laminæ. A gradual dilatation of the wound margins is carried on until the lateral displacement is considered sufficient and then a hard rubber prosthesis is inserted in the opening to prevent approximation of the borders. This plug is left in place until a solid fixation has taken place. The plastic closure of the wound is done only after the prosthesis is removed, and the opening is blocked for a sufficient period of time to prove that the parts are permanently ankylosed and the cords remain separated.

His first case was operated on four years ago and the second two years ago. Both patients lead the usual mode of life, breathe normally and have a harsh but comprehensive voice.

SCIARRETTA.

EAR

Plasmacytoma of the Petrous Temporal Bone and Base of Skull.

Cappell, D. F., and Mathers, R. P. (Dundee), J. of Laryng. and Otol., 50:340 (May), 1935.

A case is recorded of a solitary plasma cell tumor involving the petrous temporal bone, mastoid process and base of skull. The growth resulted in extensive destruction of the bony tissues, but did not invade the brain. Complete paralysis of the VIth and VIIth cranial nerves was produced, and there were also symptoms due to pressure upon the IXth and Xth and XIth nerves in the jugular foramen and to direct pressure upon the pharyngeal wall. No record of a similar case has been found in the literature.

GOLDSMITH-MCLEOD.

The Physics of the Human Labyrinth Considered as the Organ of the Equilibrium Sense: The Nystagmus Produced by the Labyrinth Stimulations (Stuzio fisico sul labirinto umano considerato comeorgano del senso dell'equilibrio: Il nistagmo provocato va sensazioni labirintiche).

Gasparini, O. (Rome), Valsalva, 10:389-440 (June), 1934.

An extremely technical article, very clearly presented, describing minutely the essential results but omitting detailed experiments.

The author examines the pressure exerted by the weight of the endolymphatic fluid on the neuro-epithelium of the semicircular canals, and by applying the approved laws of physics he succeeds in obtaining its exact measurements.

He studies how this pressure changes in the various positions assumed by the head, formulating some rules from which he deduces that the endolymphatic pressure is to be considered as the only stimulating factor for the function of the labyrinth as the organ of equilibrium.

The neuro-epithelia of the superior and lateral semicircular canals respond to the reflexes induced by the lateral motions of the head, while those of the posterior semicircular canals regulate the forward or backward movements.

According to his new conception the organ of equilibrium is not any longer represented by the vestibular portion alone but by the entire labyrinth (including the scala media).

Gasparini examines all the phenomena which are observed during an examination of the labyrinth and very minutely analyzes each one according to his newly formulated laws. He further criticizes

the accepted theory of the endolymphatic current which explains the direction and character of the nystagmus produced by labyrinthine stimuli.

The author concludes by explaining the characteristic movements of the eyes and defines this ocular reflex as a contra-rotation in respect to the false interpretation of the head movements which are produced by artificial stimulations.

SCIARRETTA.

The Gradenigo Syndrome (Sindrome de Gradenigo).

Tato, J. M. (Buenos Aires), Frascoli y Bindi Pub., 1935, p. 99.

This excellent monograph includes a critical review of forty-three reported cases with accurate roentgenographic reports, all showing petrous tip changes. The author reports several in which lipiodol reached the tip on postoperative injection. Because 12 per cent mortality has been reported he thinks that a reserved prognosis should always be made.

FENTON.

The Otosclerosis Problem: Including Reports of Two Cases Examined Pathologically.

Gray, Albert A. (London), J. of Laryng. and Otol., 49:629 (Oct.), 1934.

The writer reports two cases of otosclerosis examined pathologically and discussses the etiology of the disease. He believes that the essential causative factor of otosclerosis is a gradually increasing defect in the vasomotor mechanism which governs the nutrition of the structures of the organ of hearing as a whole. The axon reflexes are included in this vasomotor mechanism, and the stimulus which excites the vasomotor mechanism is sound and sound alone. Consequently the vestibular apparatus and the semicircular canals are unaffected in otosclerosis.

There is no evidence whatever of any defect in any of the endocrine glands or their secretions in otosclerosis. Neither is there any evidence of any defect in the bone metabolism of the body. On the contrary the subjects of otosclerosis are, apart from their deafness, perfectly normal individuals with ordinary average health.

The deafness of otosclerosis bears very little relationship to the extent of the disease in the bone and may be very severe when the stapes is hardly fixed at all. It is to a large extent functional and the result of the insufficient supply of blood to all the nerve structures concerned in the perception of sound. The preponderance of women as subjects of otosclerosis is the result of the greater instability of their

vasomotor system and the more frequent disturbances to which it is exposed.

The extent of the change in the bone bears very little relationship to the character of the disease. It appears to depend upon the age of onset of the disease. The earlier the time at which the otosclerosis begins, the more extensive will the bone lesion become. The changes in the bone show a remarkable bilateral symmetry, even to minute details. This symmetrical distribution is readily explained by the writer's view of the causative factor of otosclerosis. The vasomotor nerves governing the nutrition of the organ of hearing are anatomically symmetrical like other nerve structures in the body. If, therefore, structural changes occur as a result of defective functioning of those nerves, such structural changes will naturally be bilaterally symmetrical in their distribution.

GOLDSMITH-MCLEOD.